

Surgery of the Aorta and its Branches

JAMES D. HARDY, M.S. (CHIRM), M.D.,
F.A.C.S.

*Professor and Chairman, Department of Surgery, University of
Mississippi Medical Center, and Surgeon in Chief to the
University Hospital; Chief Surgical Consultant
to the Veterans Administration Hos-
pital, Jackson, Mississippi*

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PREFACE

This monograph came about in the following way. The author had agreed to prepare for the *American Practitioner and Digest of Treatment* a series of articles dealing with acquired diseases of the aorta and its branches. The response to the appearance of the first two installments was such that the publishers suggested eventual presentation of the series in book form. By careful planning the amount of overlap among the seven articles was reduced to the minimum consistent with clarity of exposition and effective teaching.

The purpose has been to examine in some detail the pathophysiology, diagnosis and management of a wide variety of arterial diseases for which surgical therapy is available. By a liberal use of illustrations both diagnostic and therapeutic techniques have been emphasized.

This book is not designed to offer minute technical details of vascular surgery. The author anticipates that the surgeon already will have developed—through basic surgical training in the clinic and the laboratory—the essential skills with which to carry out the technical objectives proposed herein. Nevertheless the numerous operative procedures depicted do serve to familiarize the physician not involved in the practice of vascular surgery with what can be accomplished and the general operative steps by which this is achieved. Diagnostic procedures are stressed throughout.

This is a particularly productive period in the history of vascular surgery. First it has been found that

"strokes" are frequently due to extracranial arterial occlusion. Corrective surgery, promptly executed, can often prevent subsequent hemiplegic attacks. Second, certain previously confusing ischemic conditions of the arm and the hand have been shown to be due to readily remediable organic lesions. Third, correctible occlusive disease of the superior mesenteric artery may produce intestinal angina and the malabsorption syndrome long before ischemic gangrene develops, renal arterial occlusion can result in hypertension, and thrombosis of the terminal aorta can produce intermittent claudication. All these types of ischemia frequently can be relieved. Fourth, aneurysms of virtually all arteries have now been successfully excised and the resulting defect bridged with a prosthesis. Fifth, the general problem of arterial embolism, though still serious, has been attacked with some success. Finally, the management of arterial trauma has been revolutionized by prompt direct repair of injured vessels.

The bibliography following most chapters is extensive. The remarkable progress made in the surgical management of arterial diseases has resulted from the efforts of interested physicians over a period of many years. The summaries which appeared in the original publications have been allowed to remain as brief synopses of the individual chapters.

Last, the author extends his warmest thanks to Miss Betty Anne Crocker, Miss Beth Weathersby and the J. B. Lippincott Company for their co-operation and assistance.

James D. Hardy, M.D.
Jackson, Mississippi
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CHAPTER 1

General Survey of Acquired Diseases, Diagnostic Measures, and Methods of Technical Management

Arterial Diseases Particularly Amenable to Surgical Therapy

IT will be the purpose in this monograph to present the salient clinical features to be considered in the diagnosis and surgical management of the more prominent lesions which may involve the aorta and its branches (Fig. 1). The following conditions will be discussed:

Part I *Brief General Survey of Acquired Diseases
Diagnostic Measures and Methods of Technical
Management*

Part II *Occlusive Disease of the Innominate Carotid Subclavian and Vertebral Arteries*

Part III *Diseases Involving the Arterial Supply to the Upper Extremities*

- a. Scalenus Anticus Syndrome
- b. Cervical Rib Syndrome
- c. Occlusive Disease of the Axillary Artery
- d. Reflex Vascular Dystrophy (Sudeck's Atrophy)
- e. Raynaud's and Other Vasospastic Diseases

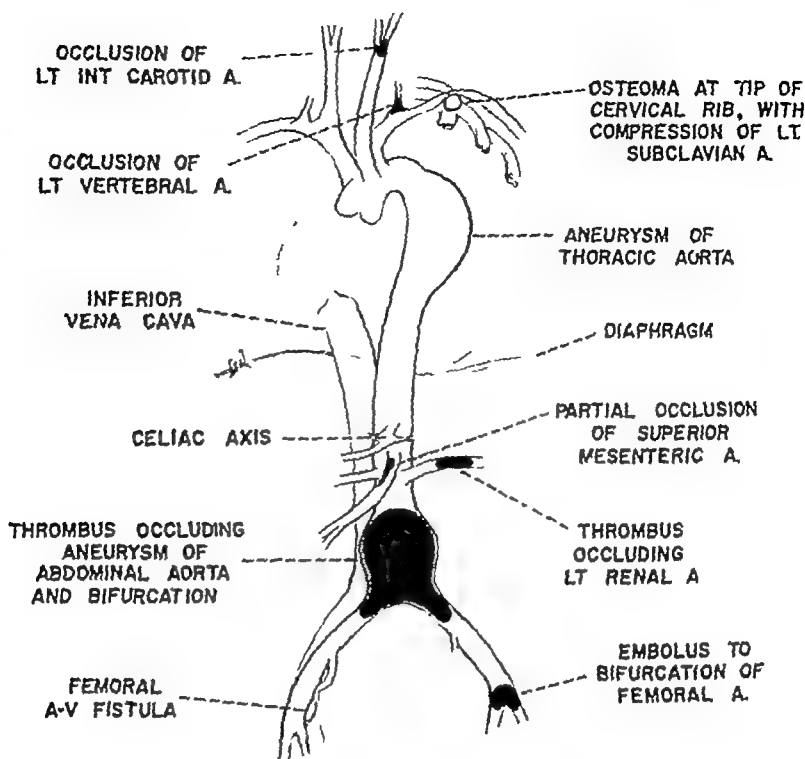


FIG 1 A number of different disease conditions of the aorta and its branches are depicted. Occlusion of the internal carotid artery or the vertebral, or both, may explain attacks of transient hemiplegia, blurring of vision or black-out spells. The cervical rib (or scalenus anticus muscle) may compress the subclavian artery to produce relative ischemia of the arm. An aneurysm of the thoracic aorta often encroaches upon surrounding nerves and other structures. Partial occlusion of the superior mesenteric artery may cause abdominal angina and a spruelike malabsorption syndrome, of the renal artery, hypertension. An aneurysm of the abdominal aorta may become completely filled with thrombus and simulate "pure" Lericq's syndrome. The fibrillating atrium or the infarcted myocardium may give rise to an embolus which occludes the femoral artery at its bifurcation. A stab or gunshot wound may result in a femoral arteriovenous fistula.

f Buerger's Disease

g Obliterative Atherosclerosis of Digital Arteries

Part IV *Occlusive Disease of the Celiac Superior Mesenteric Renal Iliac Femoral and More Distal Arteries*

Part V *Aneurysms*

Part VI *Embolism*

Part VII *Traumatic Injuries*

a Laceration (Simple)

b Loss of Arterial Substance

c Thrombosis

d Pulsating Hematoma (False Aneurysm)

e Arteriovenous Fistula

f Causalgia

There are of course additional diseases which affect arteries and in the management of some of these surgery can prove beneficial. However it is in the treatment of the conditions listed above that surgical intervention is most valuable.

By and large the adverse physiologic effects produced by disease involving an artery are those due to *spasm* (e.g. Raynaud's disease) *compression* (e.g. cervical rib syndrome) *enlargement* with rupture or compression of surrounding structures (e.g. aneurysm) *occlusion* (e.g. embolus or thrombosis) or *injury* (e.g. loss of substance or creation of an arteriovenous fistula). In few other clinical situations is a practical familiarity with the underlying pathophysiology more promptly rewarding than it is in the diagnosis and management of arterial lesions.

DIAGNOSIS OF ARTERIAL DISEASE

If one were to be allowed only two means of evaluating major arterial disease, he would be wise to choose the *clinical evaluation* and the *arteriogram*. For with the history and physical examination to suggest the presence and general distribution—and with the arteriogram to identify more precisely the level—of arterial disease, the vascular surgeon could achieve a satisfactory surgical result in the majority of cases. What, then, are some of the clinical findings which may clearly indicate the presence and the probable nature of arterial disease?

The History

General Considerations The history which the patient gives will usually depend upon the location of the arterial disease, regardless of the structural form which this disease may have taken, and the complaints will also be dependent upon whether ischemia of the tissues distal to the arterial lesion exists. For example, occlusion of the great vessels of the arch of the aorta, or of the vertebral arteries as they leave the subclavian arteries, will result in certain signs of central nervous system ischemia. Any person who has evidence of transient hemiplegia, visual field defects, aphasia, or dizzy spells should be considered as possibly having partial or complete occlusion of one or more of the four major arteries which supply the brain.

Gangrene of the tip of the finger may reflect Buerger's disease with obliteration of the small digital arteries, or it may reflect segmental occlusion of the subclavian or axillary artery by atherosclerosis.

Both types of lesions often respond quite satisfactorily to proper operative measures. Partial occlusion of the superior mesenteric artery may result in abdominal angina and may produce a malabsorption syndrome which results from inadequate arterial perfusion of the small bowel. Partial occlusion of a renal artery may cause hypertension. Finally perhaps the most common site of occlusion other than in the heart or the brain is in the arteries supplying the legs.

Pain. Except in brain lesions pain is the most characteristic symptom of arterial disease. The pain is usually due to inadequate arterial perfusion of the tissues supplied by the artery in question though the discomfort associated with an aneurysm is often produced by compression of surrounding structures. The time of onset of pain due to ischemia often bears a close relationship to exercise of the musculature involved. A most characteristic type of pain associated with relative ischemia of the legs is that of *intermittent claudication* described in man by Charcot. Initially the patient may notice only fatigue or cramps in the musculature distal to the site of arterial occlusion but this is soon followed weeks, months or even years later by actual pain which becomes incapacitating unless he stops to rest. Should the increasingly severe pain go unheeded the leg will become paralyzed due to inadequate blood supply and will collapse. *Rest pain* is that which occurs even at rest often being more severe at night. It usually occurs late in progressive ischemia and once the patient has developed this symptom gangrene may usually be anticipated unless successful surgery can restore a more adequate blood flow to the part supplied. Pain

due to arterial ischemia is often erroneously ascribed to a nerve lesion, and the patient frequently has visited a neurologist or neurosurgeon before the correct diagnosis is suspected and an arteriogram discloses occlusive disease of the artery involved

Physical Examination

Additional and more detailed points to be covered in the course of taking the history will be presented in association with consideration of specific arterial lesions later in this book. It is now in order to mention a few of the physical findings which may suggest the presence and identity of arterial disease.

Inspection The color of an extremity often reflects significant information regarding the state of the circulation. The color of the skin, disregarding various types of skin pigmentation, is usually due to the amount and color of the blood circulating through it. The less the capillary blood flow, the greater will be the relative amount of reduced hemoglobin in the blood which does traverse and leave the part. Thus the extremity with a reduced blood flow will often have a dull red color, especially when it is dependent, and it assumes undue pallor when it is elevated above the level of the heart. In both the fingers and the toes one may gain the mistaken impression that a redness (rubor) is due to infection when, in fact, it but reflects the inadequate arterial blood flow through the part. To incise such a toe or finger (a "false felon") for "drainage" may result in gangrene or at least prolonged failure of the wound to heal, for it takes less blood supply to maintain viability of untraumatized skin than it does to heal a wound. In comparing the

appearance of hands or feet the paired extremities of the patient should be examined simultaneously—dependent at the elevation of the heart and raised. It is helpful to contrast changes in the patient's hands with those of the examiner in a similar position. The extremity whose arterial inflow has been completely occluded has a marked pallor.

On inspection one also notes evidence of chronic arterial disease as reflected by trophic changes which include thinning of the skin with a shiny appearance, reduction in the volume of flesh (particularly of the pulp of the toes and fingers), loss of hair and deformity of the toenails or fingernails.

Palpation. Having discovered historical evidence of arterial disease and having visualized various of the stigmata given above, one next palpates the part. The involved extremity may be cooler than the opposite one. Or the more distal portion of the involved member may be cooler than the more proximal portion—though the feet and hands normally exhibit a lower temperature than does the trunk. Localized heat may reflect the presence of an arteriovenous fistula or hemangioma. The absence of pulsations in the radial, carotid, femoral, dorsalis pedis or posterior tibial artery reflects significant disease in most instances. If the carotid pulse is present bilaterally, one may conclude that the innominate and the left common carotid arteries are not thrombosed at their origin from the aorta. If the right carotid pulse is present but the right radial pulse is absent, the occlusion is distal to the innominate artery. Occlusion of the vertebral arteries may produce cerebral ischemia even when both internal carotids are patent. How

ever, pulsations in a common carotid artery do not rule out occlusion of the corresponding internal carotid vessel (Fig 1) Finally, aneurysms of the abdominal aorta and other accessible arteries may be detected by palpation in many or most instances

In concluding these few general remarks about certain of the more prominent features to be noted in the clinical evaluation of the patient with suspected arterial disease, it is desirable to emphasize again the many and varied clinical findings which may be encountered, since disease of any major artery throughout the body can produce symptoms peculiar to the organ rendered ischemic

Auscultation. When partial occlusion, an aneurysm, or an arteriovenous fistula is suspected, additional confirmatory evidence may be obtained by noting on auscultation the character of the bruit, which may be associated with a palpable thrill Partially occluded arteries often exhibit a systolic bruit, and occasionally a bruit may be present in both systole and diastole. Thus the presence of a bruit over a carotid artery frequently indicates partial occlusion at the carotid bifurcation The arteriovenous fistula characteristically produces a continuous bruit

The appropriate use of the sphygmomanometer affords much useful information regarding arterial pulsations and pressures

Additional Methods of Investigation

The Arteriogram Once the clinical examination has indicated disease of an artery, it is possible in most instances to obtain confirmatory evidence and precise localization with an arteriogram It may

demonstrate occlusion of an internal carotid or a vertebral artery or both to explain clinical evidence of brain ischemia. It can be used to disclose an aneurysm of the thoracic aorta. Traumatic injury to an artery of the arm or leg can be localized. Thrombosis of the superior mesenteric artery to clarify a possible cause of abdominal pain or of a renal artery to explain hypertension can be identified. Thus the value of angiography is second only to careful clinical evaluation as a means of diagnosing arterial lesions.

The arteriogram is usually performed using an iodine-containing compound. Hypaque and Urokon being the materials most commonly employed at the present time. Various volumes and concentrations are used depending upon the artery to be visualized. In general the concentration ranges from 35 to 70 per cent approximately 20 ml is injected abruptly and serial films then taken rapidly to study the vascular bed. More medium is required to visualize the aorta by angiocardiology—that is the injection of the radiopaque material into an antecubital vein and have it traverse the heart and lungs and then enter the aorta—than is required to visualize the femoral artery and its major branches in an already ischemic lower extremity. Although numerous refinements exist for the more precise and facile performance of an arteriogram under different circumstances, almost any syringe and needle will serve for the performance of most arteriograms when the vessel is surgically exposed or is immediately beneath the skin. Arteriograms of the extremities are more satisfactorily carried out under anesthesia since the radiopaque materials are quite irritating. If an anesthetic agent is not used

the patient often moves the extremity while the films are being taken and the entire radiologic study can be nullified. By temporarily compressing the main arterial trunk proximal to the point of injection, one can often achieve adequate contrast of the arterial bed in an ischemic extremity with a small amount of medium, minimizing the risk of distal vascular thrombosis and tissue necrosis.

The indications for arteriography will be considered further in connection with the management of specific lesions, but it should be stressed here that the arteriogram can produce definite and at times very serious complications,¹ among which are the following: (1) general collapse due to sensitivity to the medium (rare), (2) right-heart failure in the presence of pulmonary hypertension, apparently due to spasm of the pulmonary vessels; (3) brain damage due to focal necrosis resulting from arteriospasm and, in the case of small vessels, probably thrombosis, (4) paraplegia due to a spinal cord injury, (5) renal shutdown due to irritation, spasm, and probably terminal thrombosis in renal vessels, (6) injury to the aortic wall, possibly in an aneurysm, with extravasation of blood and/or radiopaque medium, (7) intestinal dysfunction due to injection of the main slug of radiopaque material into the superior mesenteric artery on aortogram, and (8) skin necrosis and actual ischemic swelling in the legs secondary to arteriograms performed in the ischemic lower extremity. Although most of these complications are relatively rare, few physicians who have used arteriograms extensively have failed to be confronted by one or the other of these untoward reactions upon occasion. The author has seen transient

brain damage in a child following angiocardiology death following retrograde aortography for coarctation in a woman with severe pulmonary hypertension some 12 hours following angiocardiology leakage of the radiopaque material around the artery with severe tissue reaction and prolonged neuralgic type pain ischemic changes in the hand transient oliguria skin necrosis in the leg following femoral arteriography and marked swelling of the leg with severe ischemic pain which subsided only after several weeks In the last instance there was no question but that the arteriography had further reduced the volume of blood flow through an already severely ischemic extremity

Therefore it is clear that arteriography though essential in many patients should be used only where definitely indicated The materials and techniques employed have steadily improved in recent years but there is still the need for anesthesia to reduce the pain of intraluminal irritation Actually a careful physical examination will often provide sufficiently precise information to permit corrective surgery without the necessity for an arteriogram In our own clinic we no longer perform an aortogram when the aneurysm of the abdominal aorta is easily palpable and we have ceased to perform an aortogram when no pulses are present in the femoral arteries We prefer to open the abdomen and thus determine from direct examination of the lower aorta and the iliac arteries the extent and nature of the disease In the case of occlusive disease we next explore the femoral arteries on either side below the inguinal ligament and at this time arteriography (Fig 2) is often performed to make



FIG 2 This arteriogram revealed segmental occlusion of the distal portion of the femoral artery as it reached the popliteal space. Note the precarious collateral circulation through which viability of the extremity distal to the occlusion is maintained.

certain of an adequate runoff in the more distal arteries, prior to completion of a bypass from the aorta to the femoral artery or below. This more conserva-

tive use of the aortogram is a direct outgrowth of our own and the reported experiences of others with complications from this procedure

The interpretation of arteriograms is usually a relatively simple problem but one should fully appreciate that in particular the aortogram may be misleading from time to time. In our own experience it has appeared to show occlusion where occlusion did not exist and it has appeared relatively normal when the intraluminal diameter of the incompletely obstructed artery had been narrowed to a point where the patient was having intermittent claudication. When the clinical evidence of arterial occlusion is unmistakable the poorly visualized artery may in fact represent incomplete occlusion of the vessel rather than a technically imperfect arteriogram. Furthermore the level of division of a traumatized artery as visualized on arteriogram may appear to be far above the actual site of damage the nonvisualized segment being filled with clot or in severe spasm."

Oscillometry **Precise Skin Temperature Measurements, and Plethysmography** These three methods more often used in physiologic investigations than in actual clinical practice represent refinements in the evaluation of phenomena which can be grossly estimated by physical examination and with the sphygmomanometer. The oscillometer is useful in recording fairly accurately for comparative purposes the relative pulsatile flow in the two legs or arms. The excursions which the needle makes on the dial with each pulsation are greater or lesser depending upon the volume of blood flow through the extremity. Of course the measurement of intra arterial pressure

with a needle and an appropriate electronic recording device is superior to either the oscillometer or the sphygmomanometer in identifying reductions in blood pressure (and presumably blood flow) through a particular artery, as compared with the opposite member or with normal values

Precise measurement of skin temperatures with a thermocouple is also but a refinement of changes which can be estimated grossly by palpation. However, temperature measurements are of definite assistance in evaluating the effect of sympathetic nerve block in raising skin temperature, prior to performance of sympathectomy if indicated on the basis of these measurements. Secondly, skin and muscle temperature measurements are useful in evaluating the benefit derived from vasodilator drugs. Thirdly, skin temperature values are helpful in comparing blood flow through adjacent digits or in comparing the two hands or legs. Nevertheless, the great advance in arterial surgery which has come about in the past ten years has emphasized the basic requirement of the re-establishment of an adequate volume of blood flow through vessels whose internal diameter has been restored virtually to normal. If anything, these advances have further minimized the value of drug therapy and frequently even of sympathectomy, since there is no adequate substitute for restoration of a normal intraluminal diameter. In other words, if the surgeon must resort to the use of a thermocouple to determine whether or not the operation has increased blood flow through a previously ischemic and cold extremity, the patient's symptoms will probably be relieved only briefly if at all.

Plethysmography * is used in much the same circumstances as is the precise measurement of skin temperatures. In effect it reflects the volume of blood flowing through the extremity or finger that is being examined. And here again if the increase in blood flow that has been achieved by operation or other means is so small that plethysmography is required to detect it the chances are that the patient has not been substantially improved to the point that prolonged relief from ischemic pain will have been provided.

Nevertheless any or all of these techniques may upon occasion aid in the selection of the patient who may be substantially benefited from sympathectomy of an arm or a leg to reduce vasospasm *per se* (e.g. Raynaud's disease) or a vasospastic element which is superimposed upon arterial occlusive disease (e.g. Buerger's disease). In some patients the reduction of this arteriospasm is sufficient to maintain viability of a leg for many months following the time when the patient might otherwise have required an amputation—and perhaps time is thus gained in which collateral circulation can develop.

METHODS OF TECHNICAL MANAGEMENT

Having indicated the diseases which more commonly affect the aorta and its various branches and having noted the major diagnostic measures that are most often employed we turn now to a brief consideration of the methods by which the arterial lesions disclosed are currently managed. In general these consist of excision with or without grafting, thromboendarterectomy and bypass (Fig. 3).

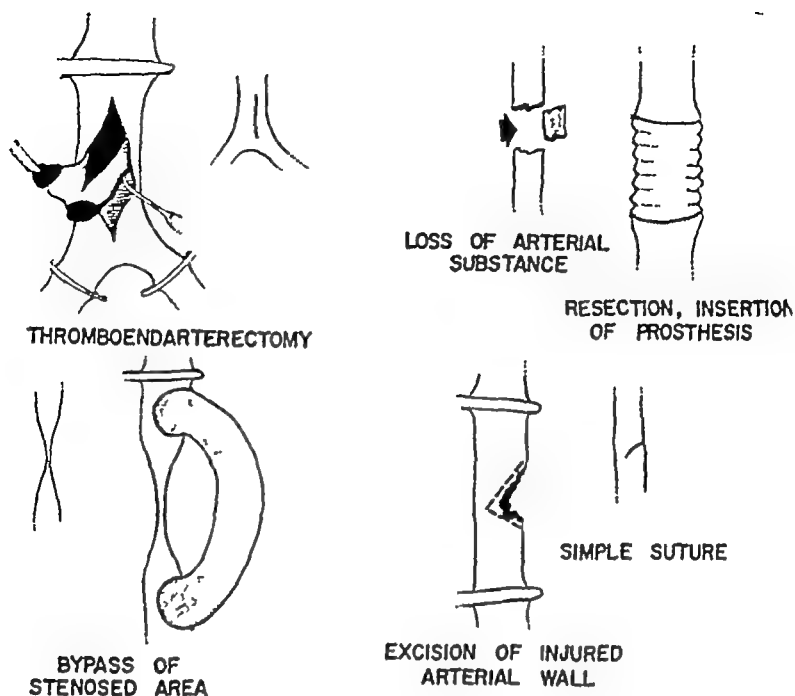


FIG 3 These several procedures illustrate the basic tactics employed in direct surgical attack upon arterial defects

Resection and Grafting

Despite the fact that Carrel³ had reported successful homotransplantation of cold preserved arterial segments as early as 1907, only during the last decade have homografts been used clinically—first by Gross and his associates⁷ in the replacement of coarctation of the aorta in 1948, and later by Dubost⁵ in 1952 for the excision and replacement of an aneurysm of the abdominal aorta. These horizons were then extended by DeBakey's group and many others, until both aneurysmal and occlusive disease of all components of the aorta and its major branches have now been resected

and grafted. However, whereas the aneurysm still must be excised and replaced with a homograft or (now far more frequently) with a synthetic prosthesis, occlusive disease of the lower aorta and iliac arteries is now rarely resected. It is managed either by thromboendarterectomy or by the use of a bypass extending from above the level of the occlusion to below it. In other circumstances, particularly in the management of traumatic arterial injuries, the defect may be debrided and sutured without the use of a graft or bypass.

Thromboendarterectomy

This procedure consists of opening the artery either transversely or longitudinally and by means of a suitable instrument, establishing a plane of cleavage between the thrombus with its firmly attached intima and the outer layers of the arterial wall. This plane of cleavage is usually readily established and the "plug" consisting of the thrombus with adherent intima and a portion of the media is freed up in its entire circumference and removed (Fig 3). If the artery is large and the thrombus is relatively short, thromboendarterectomy constitutes a very satisfactory procedure for re-establishing patency and restoring an adequate pulsatile blood flow. This technic is particularly applicable to certain lesions of the carotid, subclavian, aortic bifurcation, common iliac, and at times of the common femoral artery. It has also been used for the management of occlusion of the renal arteries and of the superior mesenteric artery. Nevertheless, one cannot determine in advance just how effective thromboendarterectomy will prove at opera-

tion If the thrombus with attached intima comes away readily and the lumen of the vessel is satisfactory with a good distal pulsatile flow, the defect in the artery is simply closed with a running suture of 4-0 or 5-0 arterial silk On the other hand, if the thrombus extends over a considerable segment of the vessel, and if the ends of the thrombus cannot be clearly visualized and meticulously excised, there is a considerable risk that thrombosis will recur Under these circumstances a bypass is to be preferred

Thromboendarterectomy is usually not a suitable procedure to employ in the management of thrombosis of very small arteries, since the diminished diameter of the vessel will all too frequently result in prompt clotting Postoperative heparinization is often employed following thromboendarterectomy upon the carotid or vertebral arteries, but we do not employ it following thromboendarterectomy performed upon the aorta or iliac vessels Considerable hemorrhage in and around the abdominal aorta has been reported following the use of heparin in connection with operations upon this vessel, and it is difficult to determine whether or not hemorrhage within the abdomen or retroperitoneal space is occurring until it is already extensive Moreover, the patients who have occlusive and/or aneurysmal disease of the lower aorta are frequently in the older age groups, and in such patients hemorrhage is all too likely to result in shock with brain damage, coronary occlusion, or oliguria In contrast, hemorrhage around the carotid or vertebral artery would usually be detected and could be adequately drained

Arterial Bypassing

The third method commonly used for direct management of a point of obstruction is arterial bypassing. This method simply consists of the establishment of a detour around the point of obstruction by means of either an arterial homograft or far more frequently at present a synthetic arterial substitute such as Dacron or Teflon. Nylon is now rarely used. The advantage of the arterial bypass is that it is more rapidly performed than is resection of a vessel as for example the lower abdominal aorta. Furthermore one does not need to free up the entire circumference of the vessel to which the bypass is to be sutured. It is necessary only to use a suitable clamp with which to exclude a portion of the wall of the vessel. An additional advantage of the bypass is that by this means collateral vessels are not disturbed. Therefore should the bypass become occluded or otherwise fail to function satisfactorily the collateral vasculature that was previously maintaining the precarious viability of the extremity is not disturbed and the patient's condition is made no worse by the operation. On the other hand, should the occluded segment be excised valuable collateral vessels might be destroyed and should the graft become occluded by clot in the postoperative period, the patient might well lose his leg—when he had walked into the hospital. Thus by the use of the bypass one does not make the patient worse and in most instances where the method is applicable he is much improved.

Use of a Patch Graft to Enlarge the Lumen of the Vessel

There is an increasing tendency to utilize a patch of synthetic material to increase the size of the lumen, perhaps following the use of thromboendarterectomy to remove an obstruction at the bifurcation of the common carotid artery. This tactic results in a circumference of the artery that is increased by the amount of the size of the patch graft, and it has been found that by such a maneuver the incidence of clotting following thromboendarterectomy is reduced. More will be said about various technics of this type in a later chapter.

Sympathectomy and Regional Sympathetic Block

The use of sympathectomy⁸ and intermittent or continuous sympathetic nerve block for arterial occlusive disease has been sharply modified and, in general, less used since a direct attack upon arterial defects became possible. There is little comparison between the improvement which results from thromboendarterectomy or arterial bypassing of an occlusion in a major artery, on the one hand, and the improvement which is realized through sympathectomy, on the other. Nevertheless, the use of sympathectomy to diminish vasospasm or even normal vascular tone, to achieve some increase in the diameter of the smaller vessels of an extremity, still has a definite place in the surgical armamentarium for the management of arterial disease. Sympathectomy may provide definite improvement in many patients whose arterial occlusion involves the small vessels of the distal portions

of the extremities where direct attack upon the arterial disease is not possible. Moreover at times severe arteriospasm which can actually threaten the viability of an extremity can be relieved by sympathetic nerve block whether or not actual sympathetic nerve section is employed. Another outstanding use of sympathectomy or sympathetic nerve block is in the management of the poorly understood causalgic pain that results from neurovascular injury it may also follow injury to an extremity through frostbite or immersion foot both of these conditions being similar with respect to the basic pathologic changes produced. Almost by definition causalgic pain is relieved by interruption of the sympathetic nerve supply to the area involved.

Use of Anticoagulants and Fibrinolysin

The use of anticoagulants in the management of major arterial lesions varies widely from one clinic to another and even in the same clinic the question of whether or not it is to be used in a given case is frequently dependent upon multiple circumstances which preclude generalizations here. More will be said about this problem in connection with the management of specific disease conditions but a common use of heparin at the present time is in connection with thromboendarterectomy and bypassing of regions involving the smaller arteries which supply the brain. I myself almost never use anticoagulants in the management of diseases of the aorta or of the vessels supplying the legs. The experience has been that if adequate technical surgery with a good 'runoff' has been achieved the anastomosis almost invariably functions

satisfactory, and the use of heparin simply adds an additional hazard which is unnecessary. Furthermore, if a technically satisfactory operation has not been performed, the use of heparin is not going to improve matters. Heparin is of course to be preferred to Dicumarol or one of its derivatives, since the heparinization can be reversed fairly promptly by the use of protamine, whereas the reversal of the Dicumarol effect on the prothrombin level by means of the infusion of vitamin K₁ oxide is not so readily reversed, should an early subsequent operation become necessary. Too, the full anticoagulant effect of Dicumarol is not achieved for from 48 to 72 hours.

There is an increasing interest in the use of fibrinolytic for the *liquefaction* and removal of recent thromboses in arteries as well as veins¹². Despite the considerable and justified skepticism regarding such usage of this enzyme *in vivo*, it is quite possible that more practical means of delivering a suitable concentration of the enzyme to the diseased segment of the vessel may be developed, in which circumstance the acknowledged ability of this enzyme to lyse a recent clot may be brought into effective therapeutic focus. Obviously the use of some such solution to remove clots in smaller vessels would be highly useful.

Homografts and Other Arterial Substitutes Further Comment

Although the early use and availability of homografts gave tremendous impetus to major arterial surgery, it was soon apparent that the other sources of material for bridging defects in arteries would be re-

quired. The reasons for this were several but the two most important had to do with the relative scarcity of homografts and with the fact that homografts were found to dilate and to rupture in some cases.⁸ Thus a large number of synthetic materials were developed and used.⁹ Of these the first one to be used was perhaps Vinyon N introduced by Voorhees, Jaretski and Blakemore.¹⁰ Thereafter the crimped Nylon prosthesis was developed by Edwards and Tapp.⁶ Recently as noted previously the materials most commonly used for bridging arterial defects have been Teflon and Dacron.

It would be erroneous to state that a truly satisfactory arterial substitute has been found. There is no question but that there will be continuous and steady improvement in the substances available for arterial replacement. However those that are in current use offer so much to the patient with for example an aneurysm that there is every reason for gratification over the development of those already available. These materials while of permanent material themselves actually become lined fairly quickly with an intima. This pseudo-intima develops from endothelial cells derived from host intima at each end of the prosthesis as well as from fibroblasts which grow through the interstices of the fabric. Moreover although these materials are not as elastic as the normal artery they do dilate with each arterial pulsation. Eventually they become encased in fibrous tissue which reinforces the tubelike prosthesis. Some do dilate and rupture.

Instruments, Suture Materials, and Methods of Suture

Instruments. Although a large variety of special vascular instruments can be effectively used upon occasion, a relatively few instruments of general utility will suffice for the general surgeon doing occasional arterial surgery. It cannot be overemphasized that gentleness and delicacy are essential to the performance of satisfactory anastomoses, particularly in the smaller vessels, and this cannot be achieved without the use of noncrushing clamps in most instances. In Figure 4 are exhibited some of the more common types of such clamps. In general the clamp either grasps the vessel gently enough so that it does not crush or "mash" through, or it has interdigitated teeth which alternate and intermesh so that the vessel is not cut in two. There are straight clamps, curved clamps, offset clamps, and various other modifications, each of which has certain advantages in different situations. Nevertheless, for most purposes the instruments shown can be used to achieve an adequate operative result. Although the basic principle employed in the manufacture of all these is that the delicate wall of the artery will not be crushed severely when the clamp is applied, in some instances almost any clamp may result in a tearing of the wall of the vessel, particularly in the atherosclerotic aorta. However, the perhaps surprising fact is that such tearing is as rare as it actually is.

Experience in the use of such clamps is readily and best achieved in the animal laboratory.

Materials. For the closure of defects in small

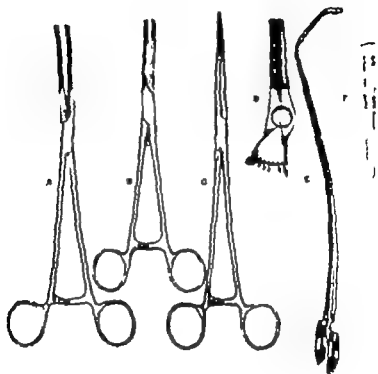


FIG. 4 (A) Curved clamp with teeth used to cross-clamp the aorta or to exclude a portion of its wall (B) Potts clamp with teeth—useful in numerous circumstances. (C) Crafoord clamp also valuable in many types of vascular and cardiac problems. (D) Bulldog clamps with which to occlude smaller vessels. (E) Satinsky clamp (F) The 4-0 arterial silk used for most arterial anastomoses. The several clamps are largely interchangeable and lend themselves to a wide range of technical requirements.

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Suture Materials For the closure of defects in small

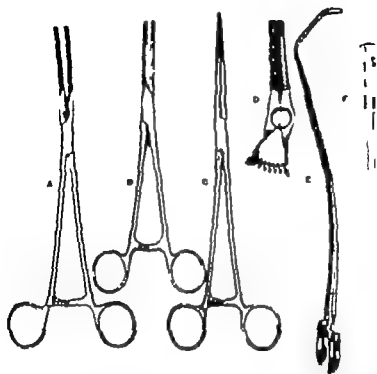


FIG 4 (A) Curved clamp with teeth used to cross-clamp the aorta or to exclude a portion of its wall (B) Potts clamp with teeth—useful in numerous circumstances. (C) Crafoord clamp also valuable in many types of vascular and cardiac problems. (D) Bulldog clamps with which to occlude smaller vessels. (E) Satinsky clamp (F) The 4-0 arterial silk used for most arterial anastomoses. The several clamps are largely interchangeable and lend themselves to a wide range of technical requirements

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Following the resection of aneurysms of the aorta, we may upon occasion use 3-0 arterial silk

Methods of Suture A decade ago most suture lines in great vessels were performed using the everting mattress suture. However, during the last few years there has been a general tendency to shift to the continuous over-and-over "baseball" stitch. In children interrupted sutures are advisable, at least for one-half the circumference—as in resection of a coarctation of the aorta where this vessel would be expected to enlarge over the years. It was shown in young pigs that the use of an interrupted suture line in the thoracic aorta resulted in a greater growth of the aorta with the growth of the pig than did a continuous suture, following excision of a segment of the thoracic aorta.¹⁰ Yet under most circumstances the simple over-and-over stitch, with the sutures being placed at approximately one millimeter intervals, is quite satisfactory for the overwhelmingly majority of vascular anastomoses. The placement of the sutures is much facilitated by the use of clamps applied and held in such a way as to take the tension off the suture line as the sutures are being placed. By the proper rotation of the clamps the anterior and posterior suture lines can be placed with almost equal facility.

Use of Hypothermia in Major Arterial Surgery

Following the advent of hypothermia as an aid in cardiovascular surgery, this technic was used extensively in many types of surgery of the aorta and its branches. Recently, though, there has been an increased use of temporary arterial bypass and a decline

in the use of hypothermia in such surgery. The reason for this is that by the use of a temporary bypass—to supply the brain for example—the patient can be maintained in a more physiologic state than would be the case with hypothermia. The onset of arterial hypotension in the normothermic patient is more promptly ascribed to the probable cause than in the case with the hypothermic patient for hypothermia itself routinely lowers the blood pressure. Nevertheless, the use of hypothermia for the correction of certain conditions such as isolated pulmonic stenosis continues to be a thoroughly satisfactory procedure.

SUMMARY

1. The surgery of acquired diseases of the aorta and its branches, diagnostic and therapeutic methods and principles have been emphasized.
2. Accurate diagnosis is most readily achieved by means of the clinical evaluation and judiciously used arteriograms.
3. The value and the hazards of arteriography have been mentioned.
4. Oscillometry, precise skin temperature measurements with the thermocouple and plethysmography are not usually essential in clinical practice despite their acknowledged value in the physiologic study of vascular problems.
5. The principal surgical techniques employed in the management of diseases of the aorta and its branches are segmental excision with or without grafting, thromboendarterectomy and bypassing of occluded channels.

6 Sympathetic nerve block and sympathectomy are less frequently employed than formerly but are still useful in carefully selected patients

7 Arterial substitutes, the use of anticoagulants, instruments and suture technics have been considered

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CHAPTER 2

Occlusive Disease of the Innominate, Carotid, Subclavian and Vertebral Arteries

STROKES AND THEIR RELATION TO THE ARTERIAL SUPPLY OF THE BRAIN

Anatomy For several decades sporadic reports have called attention to the relation between occlusion of various of the arteries supplying the brain and the onset of neurologic symptoms and signs commonly referred to as representing "strokes". However, such findings did not receive wide attention until recently. It is now accepted that many strokes are caused by cerebral ischemia resulting from partial or complete extracranial occlusion of the arteries to the brain.

The arteries arising from the aortic arch ultimately provide four major vessels, the two internal carotid arteries and the two vertebral arteries. The former join the Circle of Willis on either side, whereas the latter fuse to form the basilar artery which enters the Circle of Willis posteriorly (Fig. 5). There is considerable cross circulation between the two carotid arteries and between these vessels and the vertebral arteries through the basilar artery. Yet the Circle of Willis is more adequately developed in some patients than in others, as is the anterior portion of the basilar

artery. Thus the clinical findings produced by extra-cranial occlusion of one or more of the carotid^{2, 3, 7, 8} or vertebral arteries are further influenced by the volume of cross circulation within the skull itself. In addition to developmental variations in the di-

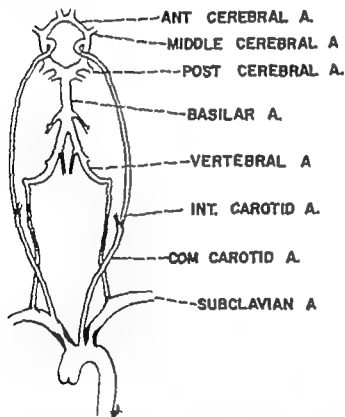


FIG. 5. Atherosclerotic occlusions tend to be segmental in distribution and to occur at bifurcations. The vertebral arteries form the important basilar artery and this vessel and the two internal carotid arteries form the Circle of Willis. In most patients there is extensive cross circulation between the two internal carotid arteries and between these vessels and the basilar artery.

anterior elements of the Circle of Willis, atherosclerotic thrombosis and occlusion of various smaller vessels such as the cerebellar arteries may occur

Sites of Extracranial Arterial Occlusion. Experience has shown that occlusion of the elements of the arterial supply to the brain commonly occurs at the origins of the vessels from the aorta, or at the bifurcation of the innominate or carotid arteries,^{28, 40, 40} or at the sites of origin of the vertebral arteries from the subclavian arteries. However, although occlusion most often occurs at a bifurcation, atherosclerotic thrombosis may occur at almost any point.²⁴ The fortunate circumstance is that many instances of occlusion are found proximally in relatively large arteries, for the larger the vessel involved the greater is the opportunity for surgical relief of the obstruction.

Perhaps the most important site of occlusion is at the bifurcation of the carotid artery,⁸ for the two internal carotid arteries carry a large part of the arterial flow to the brain. Nevertheless, Hutchinson and Yates²⁰ found that, in patients dying of cerebrovascular accidents, almost 40 per cent had insufficiency of the basilar artery resulting from obstructive lesions in the various segments of the vertebral arteries. Simultaneous occlusions of the internal carotid and vertebral arteries, of varying severity, were found in approximately a third of the cases.

The practical conclusion to be derived from these data is that, in the patient who is being investigated for ischemia of the brain, all major arteries contributing to cerebral blood flow should be evaluated by physical examination and appropriate arteriograms.

Pulseless Disease

Special mention should be accorded a condition which may affect the vessels of the aortic arch though the atherosclerotic mechanism of occlusion is perhaps little different from that which occurs elsewhere.^{2, 3, 13, 14, 25, 26, 31} The essentials of the clinical picture were recorded by Broadbent⁴ in 1875. In 1908 Takayasu⁴⁵ a Japanese ophthalmologist reported a syndrome characterized by occlusion of the main vessels arising from the aortic arch. Martorell Otlet and Fabre³⁴ described in 1914 an obliteration of the supra aortic trunks and the condition was then referred to as Martorell's syndrome. Shimizu and Sano⁴⁶ dramatized the syndrome in 1951 by terming it pulseless disease. In a review of pulseless disease published in 1952, Cacamise and Whitman⁵ reported 1 case and collected 58 additional case studies previously reported in the Japanese literature. Ross and McKusick⁴⁸ characterized this group of arterial occlusions as the aortic arch syndrome and Kalmansohn and Kalmansohn³¹ termed it thrombotic obliteration of the branches of the aortic arch.

In summary the various expressions that have been used to denote occlusion of the branches of the aortic arch include Takayasu's disease, pulseless disease, aortic arch syndrome, Martorell's syndrome, reversed coarctation (blood pressure low in the arms and much higher in the legs) and chronic subclavian-carotid obstruction syndrome.

The major signs and symptoms have to do with inadequate arterial flow to the brain. Nevertheless in the occasional patient clinical findings leading to a

diagnosis of syringomyelia or other nervous system disease have long preceded symptoms obviously due to ischemia. This prompts one to wonder whether or not certain neurologic diseases of "obscure origin" may not be due in part to selective vascular insufficiency.

One conspicuous feature of pulseless disease is the relative frequency with which it is found in young or middle-aged women, in contradistinction to the relatively infrequent involvement of arteries elsewhere in women prior to the onset of the menopause. Several causes have been proposed, among them syphilitic aortitis, nonspecific arteritis, atheromatosis, and others. However, the etiology in most instances is open to question and, since cases are now being found in all races and age groups, one suspects that atherosclerosis per se is a prominent causative factor.

The Natural Course and Prognosis of Strokes

A knowledge of the probabilities following a cerebrovascular occlusion is helpful in the management of the condition. Robinson and his associates⁴⁴ examined the records of 1018 patients with cerebral thrombosis admitted to three major Worcester (Massachusetts) hospitals during the years of 1947 through 1956. The initial attack was fatal in 21 per cent of cases. Of the 737 patients who survived the first episode of thrombosis, 50 per cent died within 41 years, while only 18 per cent of a comparable sample of the general population died within that time. The principal cause of subsequent mortality was recurrent vascular disease, which accounted for 85 per cent of all deaths.

These data underscore the value of prompt diagnostic angiography as soon as neurologic symptoms have appeared to permit immediate surgical intervention where applicable.

Fortunately the initial stroke may be extremely mild and transient with no residual neurologic deficit. And it is in such patients that the greatest rewards attend surgical therapy. Using general approximations one may assume that in about 35 per cent of patients the initial stroke will be sudden and massive and surgery has benefited few of this group. In about 25 per cent the initial effects of the stroke are not catastrophic but the neurologic deficit is progressive over the next few hours or days until marked disability obtains. Such progression may be due to propagation of the initial thrombus. Early surgical intervention may prove to be of considerable value in this group. Finally but exceedingly important in about 40 per cent of cases the initial stroke is mild and fleeting; this affords ample warning and provides time in which precise anatomic diagnosis and surgical correction can be carried out to prevent subsequent episodes in many patients.

DIAGNOSIS OF OCCLUSION OF ARTERIES SUPPLYING THE BRAIN

For effective surgical intervention it is necessary to localize precisely the level or levels of arterial occlusion. This is accomplished with data obtained from the history, physical examination, certain additional measurements, and arteriograms.

The History

The symptoms experienced by the patient are related to the several parts of the body supplied by the arteries in question¹⁰ Since the major trunks involved include the carotid and subclavian vessels, the body areas primarily affected are the brain, eyes, and the upper extremities; ulcerations and other ischemic lesions of the face have been reported

The central nervous system manifestations range from mild symptoms of headache, dizziness, and fainting spells to more severe epileptiform seizures which may be followed by major neurologic deficits such as hemiplegia Prominent may be periods of temporary partial or complete blindness, often associated with the previously mentioned vertigo Speech disorders are frequent In the past these fainting spells were probably considered to be the result of a "carotid sinus syndrome" in many instances When there is thrombotic occlusion of the internal carotid artery on one side, compression of the carotid on the opposite side may produce such brain ischemia that marked hypotension, fainting, and epileptiform convulsions result Marked electroencephalographic changes, along with typical neurologic symptoms, may be produced in such patients by change of position on a tilt table³⁷

Subclavian artery occlusion, which may have reduced the flow through the corresponding vertebral artery (Fig 5), results in ischemic changes that may produce intermittent claudication of the arm, or secondary Raynaud's phenomena in the fingers with vasospasm, cyanosis, trophic changes, and even mild ulceration

Physical Examination

Once the symptoms have suggested focal ischemia of the brain physical examination may afford additional evidence of arterial occlusion. If the pulsations in the common carotid arteries are of good quality and the brachial arterial blood pressure and radial pulses are normal in each arm occlusion at the aortic arch (Fig 5) is essentially excluded. If abnormal pressures or absent pulsations are found an aortogram is indicated.

Thrombosis of the internal carotid and vertebral arteries is not readily excluded by physical examination and arteriograms are essential. Nevertheless partial occlusion of either the internal or external carotid, or of a subclavian or vertebral artery may be suspected if a *bruit*^{12, 13} and *thrill* are detected over the carotid bulb or in the supraclavicular fossa medially respectively. This partial occlusion may be sufficient to produce basilar arterial insufficiency upon occasion with transient visual disturbances, dizziness or fainting spells. These attacks may be precipitated by sudden changes in posture and again may be associated with electroencephalographic alterations.

In addition to simple blood pressure measurements in the two arms *oscillometry* and *finger plethysmography* may be helpful in detecting differences in blood flow through the arteries of the upper extremity.

Ophthalmodynamometry. Wood and Toole¹⁴ have emphasized the value of ophthalmodynamometry for the identification of occlusion of the internal carotid artery on the same side. In brief a relative pressure measurement is made in the central artery of the retina (a branch of the ophthalmic artery Fig 6) by gradual

compression of the globe and the demonstration of obliteration of flow in the central artery of the retina at a lower level than normal. For example, as the pressure is increased, pulsations usually appear in the artery at between 20 and 70 grams of pressure and usually cease at between 60 and 130 grams of pressure. In five cases of proved occlusion of the internal carotid artery the pressure in the central artery was found to be subnormal. This procedure is considered to be somewhat safer as a test than is the use of digital compression of the unaffected carotid artery, since such compression may set off a convulsion with a marked drop in blood pressure and possible unfavorable sequelae such as hemiplegia.

In examining for the possible presence of occlusive

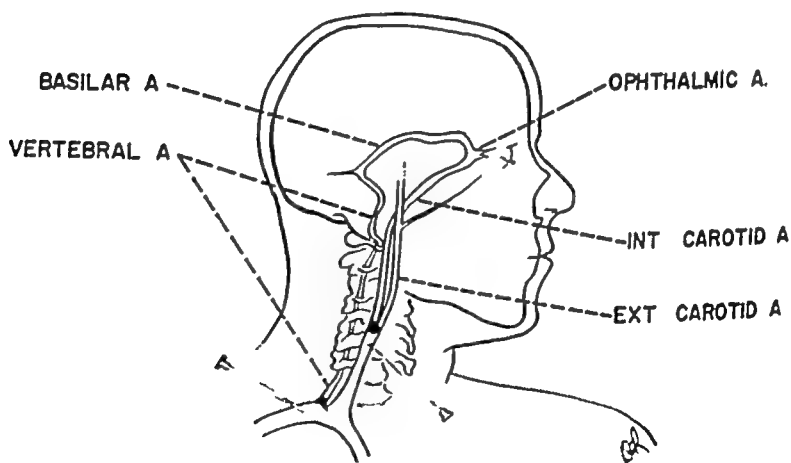


FIG. 6 The fact that the ophthalmic artery supplies the retina forms the basis for ophthalmodynamometry in the diagnosis of reduced pressure in the Circle of Willis. Note sites of injection for carotid and vertebral (through subclavian) arteriography.

disease of the arteries supplying the brain it is always important to rule out other lesions. Both vascular insufficiency and brain tumor may be found in the same patient upon occasion and of course the one may mimic the other.²⁸

Angiography^{2 10 11 20 21 22}

Arteriography for the localization of intracranial tumors was first reported by Moniz² in 1927. The technic was extended by Robb and Steinburg¹⁰ in 1939 when they reported visualization of the chambers of the heart, pulmonary circulation and the aortic arch with its great vessels in man. Visualization of the vertebral arteries is achieved by injecting the radiopaque medium into the subclavian artery on either side (Fig. 6) or directly into the vertebrals themselves.

Selection of Arteriographic Approach. Absence of pulsations in the subclavian or common carotid arteries requires angiographic visualization of the aortic arch. This may be achieved in a variety of ways including complete angiocardiology through an antecubital vein or by retrograde injection through a cervical branch of the aorta or by passage of a cardiac catheter retrograde through a femoral artery. If the common carotid pulses are present, carotid arteriograms are required to exclude occlusion of an internal carotid at the level of the carotid bifurcation (Fig. 7). In addition the vertebral arteries should be visualized by means of subclavian arteriograms. Since at present the intralaminar portion of the vertebral artery is not accessible to surgical attack, direct vertebral arteriograms add little to the end results of



FIG 7 Carotid arteriogram. Note almost complete occlusion of the left internal carotid artery by an atherosclerotic plaque. The patient had experienced several episodes of visual disturbances accompanied by vertigo.

surgery directed toward the relief of occlusive disease. On the other hand, the more distal portions of the

vertebral arteries as well as the basilar artery are more vividly outlined when the radiopaque medium is injected directly into the vertebral artery instead of into the subclavian and tumors of the posterior fossa or occlusion of the basilar artery is better demonstrated by direct vertebral arteriography.

Hazards of Cerebral Arteriography Important and highly valuable as is cerebral arteriography it is attended by very definite hazards. The smallest amount of material in the lowest practicable concentration should be employed for visualization of the vessel in question. First the infiltration of the commonly used materials such as Hypaque or Urokon around the vessel may result in marked pain and inflammation often associated with vasospasm. The brachial plexus is of course adjacent to the subclavian artery. Second the injection of the material into the carotid artery for example frequently causes the patient pain and discomfort and may produce considerable vasospasm in the smaller vessels of the brain. Such vasospasm with the attendant inflammatory reaction may result in thrombosis with additional impairment of the blood supply to the part. Brain damage secondary to carotid arteriography is not particularly rare.

MANAGEMENT OF OCCLUSIVE DISEASE OF ARTERIES SUPPLYING THE BRAIN

It was pointed out earlier that approximately 35 per cent of strokes are sudden massive and permanent about 25 per cent are progressive and about 40 per cent are transient. It is those patients with minor and transient strokes who are most likely to be benefited by corrective surgery where the etiology of the stroke

is that of partial or complete occlusion of one or more of the major arteries supplying the brain. There are of course medical measures which can be instituted in those patients in whom surgery is contraindicated for one reason or another. These include general supportive treatment, vasodilator drugs, and anticoagulant therapy—in addition to cervical sympathetic block. Associated vasospasm may occasionally play some role in the total effect of cerebral thrombosis in producing cerebral ischemia. Nevertheless, it was not until reconstructive arterial surgery became available for the management of brain ischemia that truly significant advances were made in the treatment of this frequently catastrophic condition. Furthermore, even patients who have had a sudden and massive stroke occasionally will improve if operation is carried out immediately. What is required is that angiography be performed promptly, and the site of occlusion identified at once. Operation is then executed at the site indicated, under local anesthesia where possible, if the occlusion is located within the thorax, light general endotracheal anesthesia is used.

Again, when the appropriate arteriogram is performed and demonstrates one specific site of partial or complete occlusion, thrombosis of other major arteries should be demonstrated or excluded. The reason for this is that, whereas partial occlusion of one internal carotid artery may have been disclosed by the initial arteriogram which was prompted by a transient stroke affecting the opposite side of the body, the other internal carotid may be completely occluded. Moreover, one or both of the vertebral arteries may be partially or completely occluded. In the event that one

internal carotid artery should prove to be completely occluded with the other one only partially occluded it will frequently be possible to perform endarterectomy or to do a bypass of the completely occluded artery first without the use of a temporary shunt or hypothermia—this being possible because of the flow afforded by the partially occluded opposite internal carotid artery and through the vertebral arteries when they are not occluded. Thereafter the partially thrombosed carotid is in turn attacked. On the other hand to attempt to occlude the partially thrombosed internal carotid artery even temporarily in the presence of complete occlusion of the opposite internal carotid artery might well result in serious brain ischemia. Whether or not a temporary bypass is required may often be determined by the reaction to temporary occlusion of the internal carotid artery on the exposed side: should the patient tend to lose consciousness or the blood pressure to fall a temporary shunt must be employed. It is not often necessary to use a shunt¹⁸ for thromboendarterectomy in the relief of occlusion at the aortic arch since collateral circulation is usually extensive. However thromboendarterectomy is usually avoided here in favor of the bypass procedure.

Operative Approaches to the Ascending Aorta and Aortic Arch, Great Vessels, Vertebral Arteries and Internal Carotid Arteries

In Figures 8 and 9 are shown incisions for exposure of the ascending aorta, aortic arch, subclavian, vertebral, axillary and internal carotid arteries. The ascending aorta for use in the insertion of a bypass prosthesis may be exposed through either a right third

intercostal space incision or through a sternal splitting incision. The latter gives excellent exposure, but it is more traumatizing to the patient than is the intercostal incision. Furthermore, the edges of the divided sternum may be the site of considerable oozing in the postoperative period, though this may be controlled with bone wax or by closure of the sternum over gel foam. Adequate exposure of the vessels of the aortic arch is best secured through a limited median sternotomy incision (Fig 9). The common carotid arteries are readily exposed through a transverse cervical incision, as is a portion of the subclavian artery on either side. The bifurcation of the common carotid artery is exposed with an incision running along the border of the sternocleidomastoid muscle in the appropriate location. The incision for exposure of the vertebral and the axillary arteries is shown in Figure 8.

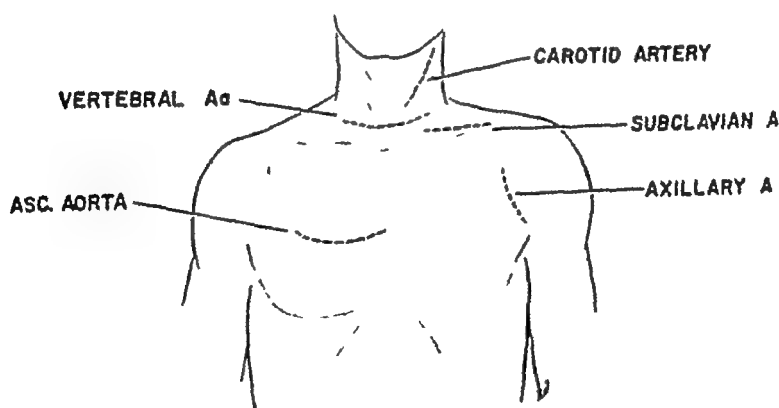


FIG 8 In addition to the incisions indicated a sternal splitting approach is used for exposure of the aortic arch. The exposure of the subclavian artery is for exposure of the axillary artery and the vertebral artery.

Frequently separate short proximal and distal incisions are adequate and one thus avoids a long incision. The prosthesis is anastomosed proximally and then a tunnel is made with the finger or a Kelly hemostat to pass the prosthesis from below to the point above at which it is to be anastomosed distal to the level of arterial occlusion.

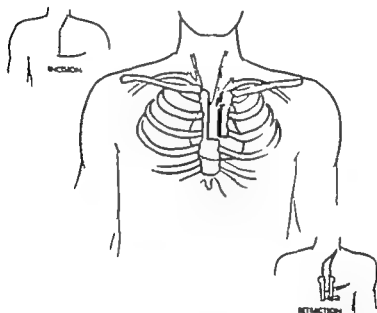


FIG. 9 The sternal splitting incision is carried into the right third interspace or the left third interspace depending upon the exposure desired. It is not often necessary to excise the medial third of the clavicle on the side where the greater exposure is desired. The sternum is reapproximated with interrupted wire and silk sutures. Oozing from the divided margins of the sternum is controlled with bone wax and gelfoam. At times electrocautery is also helpful though prolonged and excessive blood loss from this source is uncommon unless hypothermia or the pump-oxygenator is used.

Aortic Arch Occlusions: Innominate, Left Common Carotid, and Left Subclavian Occlusions of the great vessels arising from the aortic arch are best managed by arterial bypass in most instances (Fig 10). A portion of the wall of the ascending aorta is temporarily excluded by means of a special clamp, and the proximal end of the prosthesis is then anastomosed to it. The distal end of the prosthesis is anastomosed to the appropriate vessel beyond the point of occlusion. This avoids the necessity for temporarily occluding the aorta at the arch, as would be the case with end

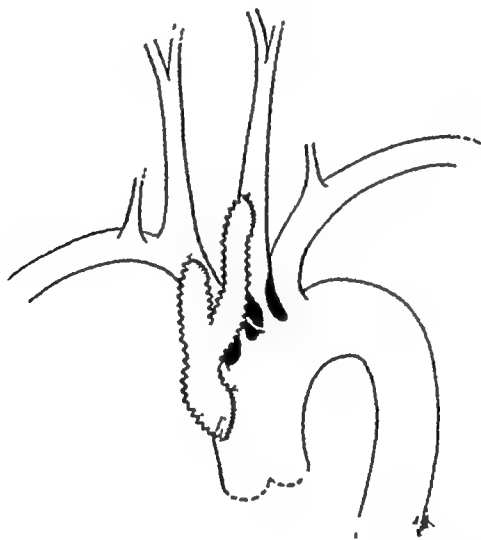


FIG. 10 Occlusive disease at the origin of the great vessels from the aortic arch is best managed with arterial bypass graft. The proximal end of the prosthesis, usually Teflon or Dacron, is anastomosed to a portion of the wall of the ascending aorta which has been temporarily excluded with a special curved noncrushing clamp. The distal end of the prosthesis is anastomosed end to side to the appropriate artery distal to the point of occlusion.

arterectomy or resection and it establishes a satisfactory pulsatile flow. The actual location of the distal anastomosis will be dependent upon the extent, location and number of vessels affected by the occlusive process. Multiple involvement of the vessels of the aortic arch is frequent and a bifurcated or even a trifurcated graft may be desirable.¹⁷ The length of the arterial segment which is occluded varies but it is often quite short and the entire bypass may be confined to the chest.

Internal Carotid Artery Occlusion^{1 7 16 18 20 21 22 23 41 42 47} If a pulsating common carotid artery on each side indicates that there is no occlusion of these vessels at their origin from the innominate artery and the aorta respectively, angiograms performed directly through the common carotid vessels may disclose occlusion of an internal carotid artery on one side (Fig. 7) or on both sides. Again the status of the vertebral arteries should also be determined by subclavian arteriograms (Fig. 6) before operative intervention is undertaken.

Assuming that complete carotid occlusion has been demonstrated on one side perhaps with partial occlusion on the other, a suitable incision is made to expose the carotid bifurcation on the side of complete occlusion. Should the occlusion attacked be incomplete the desirability of the use of a temporary bypass (Figs. 11 and 12) extending from the common carotid artery to the internal carotid artery distal to the level of occlusion at the bifurcation must be considered. As indicated above, since the operation is preferably done under local anesthesia any central effect of the temporary occlusion of this limited blood flow will be

noted immediately and a bypass can be used if the patient exhibits symptoms referable to brain ischemia. Whether or not a bypass is required in the given case, the occlusion of the internal carotid artery may be managed by either endarterectomy (Fig 13) or bypass graft. The majority of such occlusions are suitable for endarterectomy and, since this represents a simple procedure, it is employed by the majority of sur-

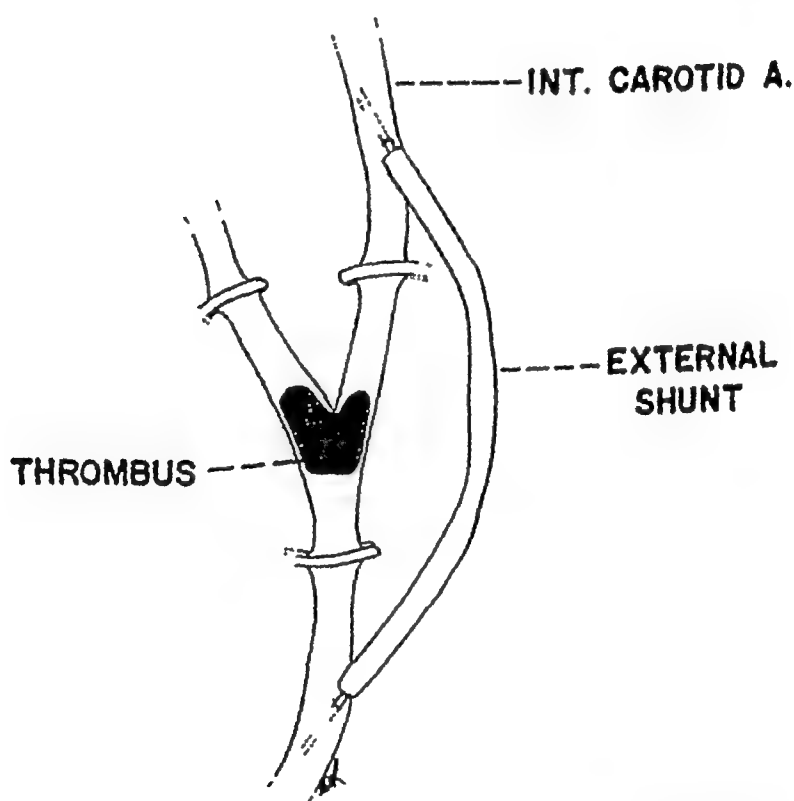


FIG 11 Thromboendarterectomy can often be performed without the use of either a shunt or hypothermia if the internal carotid artery has been completely occluded. The external shunt is readily applicable in most circumstances where a shunt is required.

geons. The endarterectomy is preferably performed through a transverse incision made in the common carotid artery just below the bifurcation. By making the incision in the larger of the three arterial branches which contribute to the bifurcation one reduces the possibility of seriously encroaching upon the lumen of the internal carotid artery when closing the arterial incision. The artery may be par-

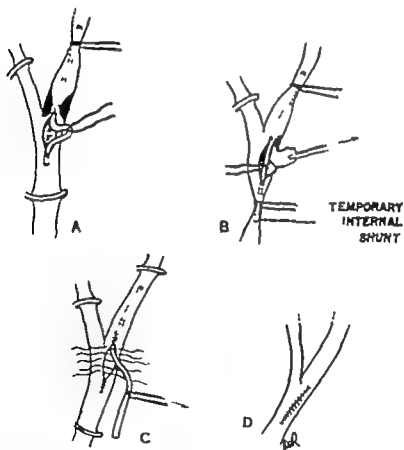


FIG 12 The internal shunt permits a greater volume of blood flow during endarterectomy than do most external shunts.

tially or completely divided with this incision (we prefer the former), and the endarterectomy carried out upon the involved segment of the common carotid artery as well as its internal and external branches. Following this the arterial defect is closed by transverse suture.

It is frequently advisable to use a patch graft (Fig 14) in the course of closing the arterial incision, particularly if it has been necessary to make an incision in the internal carotid artery.¹⁰ This affords an in

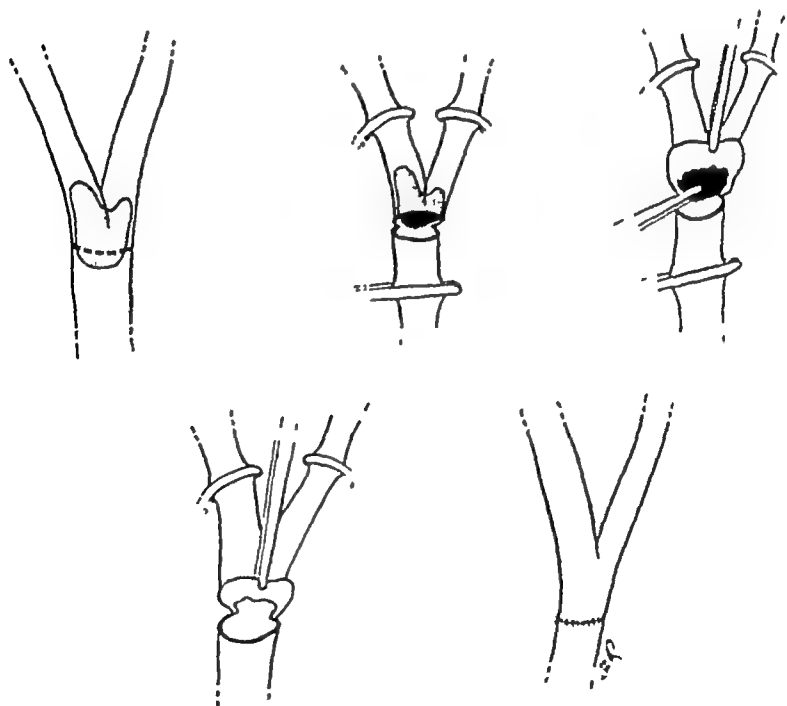


FIG. 13. Thromboendarterectomy is currently the procedure of choice for the management of most instances of occlusion at the bifurcation of the carotid artery. Where it is not applicable because of the length of the segment involved in the occlusive process, a bypass graft may be employed.

crease in the size of the circumference of the internal carotid artery roughly equivalent to the size of the small patch graft employed. By this tactic a reduction in the intraluminal diameter of the vessel is avoided.

Should an endarterectomy prove not to be feasible in the given case perhaps because of thrombosis of a relatively long segment of the artery, an arterial bypass graft can be inserted from the common carotid artery below to a point distal to the level of occlusion of the internal carotid artery. In general the bypass graft should be of greater diameter than is the internal carotid artery to which it is being anastomosed in an end-to-side fashion.

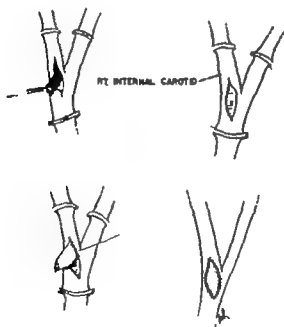


FIG. 14. A patch graft permits one to avoid encroachment upon the lumen of the carotid vessel in closing the incision.

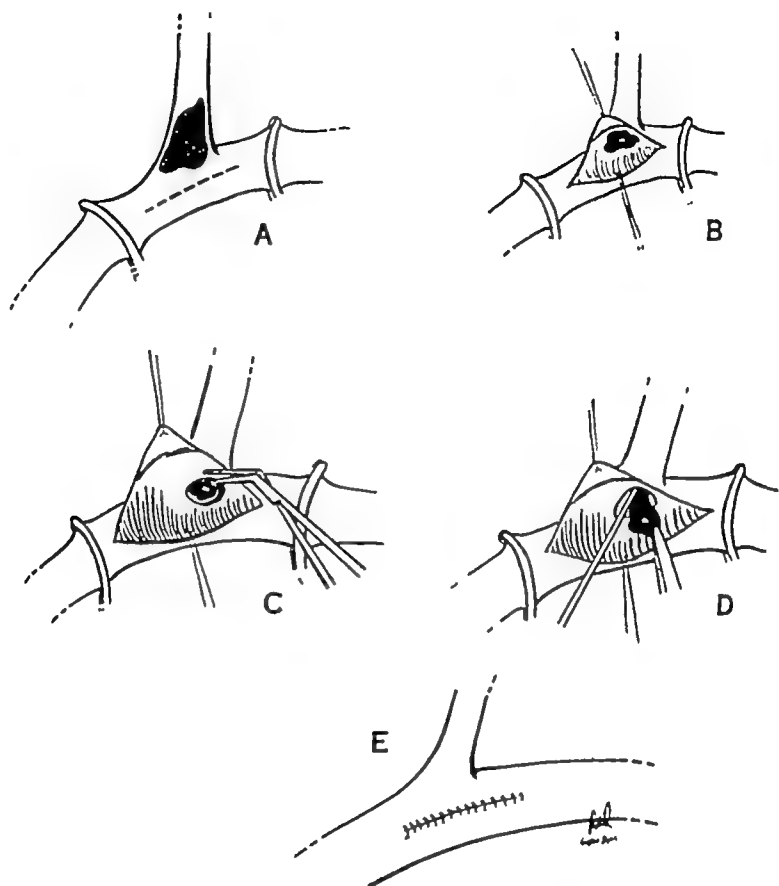


FIG. 15 Since segmental thrombosis of the vertebral arteries most often occurs at their origin from the subclavian arteries, vertebral thromboendarterectomy is best performed through an incision in the much larger subclavian artery.¹⁶ If the occluded segment is long, however, a bypass graft from the subclavian artery distally to the vertebral artery beyond the level of occlusion may be applicable. The portion of the vertebral artery which courses through the cervical transverse processes (Fig. 6) is not readily accessible to surgical attack.

Vertebral Artery Occlusion. Thromboses involving the vertebral arteries are usually short and located at or near the subclavian arteries.^{6, 16} Once complete occlusion has occurred however the thrombus may propagate. In most instances it is possible to find a patent segment proximal to the anastomotic portion of the vertebral artery (Fig 6). It is not uncommon for both vertebral arteries to be occluded but the successful endarterectomy or bypass of one occluded vessel may supply sufficient blood flow to the basilar artery and its branches to permit adequate oxygenation of the brain. Since an incision in the relatively small vertebral artery with longitudinal closure carries with it a considerable hazard of constriction of the lumen of this vessel and subsequent thrombosis the endarterectomy is often best performed through an incision made in the subclavian artery opposite the origin of the vertebral artery (Fig 15).¹⁶ If the occlusion of the first part of the vertebral artery is extensive but is still associated with a patent segment proximal to the vessel's entrance into the transverse processes of the cervical vertebrae a bypass is employed both the subclavian and vertebral arteries usually being exposed through a supraclavicular incision in the neck (Fig 8). Occasionally however a vertebral artery arises from the subclavian artery in the mediastinum and under these circumstances a combined cervico-thoracic incision with splitting of the sternum to the level of the third interspace is employed (Fig 9).

Additional Considerations During Surgery

It is extremely helpful to maintain the patient in a state of consciousness wherever possible. Hypoten

sion is scrupulously avoided by the use of adequate blood transfusion and vasopressor agents as required—for even brief drops in blood pressure may result in hemiplegia. DeBakey and his associates¹⁶ have recommended that the blood pressure be elevated in peripheral arteries to perhaps 200 millimeters of mercury during the period of occlusion of the internal carotid on the side where the operation is being performed. This is accomplished by using a continuous intravenous drip of dilute neosynephrine solution to increase blood flow through collateral channels in order to prevent a sudden drop in blood pressure from blood loss or noxious reflexes.

Results of Surgery

In suitable cases the results of surgery have been most favorable. Where the level of arterial occlusion is susceptible to endarterectomy or arterial bypass graft, with restoration of pulsatile flow to the distal arteries continuing to the brain, the patient's episodes of transient cerebral symptoms have been relieved completely. Once again, it is extremely important to diagnose arterial occlusion before a severe stroke has occurred. Patients who have had transient central neurologic phenomena respond far more favorably to careful reconstructive surgery upon the arteries supplying the brain than do those who have had a major cerebrovascular episode with perhaps permanent neurologic deficit. Nevertheless, even those who have had major strokes should have the benefit of immediate arteriography for, should a block susceptible to surgical correction be demonstrated, prompt operative in-

intervention may result in the salvage of a number of such patients

SUMMARY AND CONCLUSIONS

1 Brain ischemia is frequently due to occlusive lesions of extracranial arteries that are readily amenable to surgical attack.

2 The great branches of the aortic arch ultimately give rise to the two internal carotid arteries and the two vertebral arteries, which form the Circle of Willis and the basilar artery respectively. Arterial insufficiency may be due to thromboses situated at any point between the aorta and the smaller intracranial branches.

3 The common sites of extracranial atherosclerotic thrombosis are found at the origin of the innominate left common carotid artery and left subclavian artery along the aortic arch at the bifurcation of the common carotid arteries and at the origin of the vertebral arteries from the subclavian arteries. When occlusion of both common carotid and both subclavian arteries occurs the syndrome is often referred to as pulseless disease.

4 In about 35 per cent of patients the initial stroke is sudden, massive, and permanent; in about 25 per cent the attack is not severe initially but is progressive and may over a period of days result in marked disability; and in about 40 per cent the initial episode is mild and transient. It is in the last group that prompt diagnosis, arteriographic localization, and surgical intervention are most rewarding.

5 The symptoms and signs of brain ischemia range from mild headache, vertigo, and fainting spells to more severe epileptiform seizures which may result in

major neurologic deficits such as hemiplegia. Temporary episodes of visual disturbances and speech disorders are common. The "carotid sinus syndrome" is now known to reflect partial or complete occlusion of an internal carotid artery in many or most instances.

6. The diagnosis of occlusion of one or more of the great vessels at their point of origin from the aorta can be made from physical examination and confirmed with aortography. Stenosis of the internal carotid or vertebral arteries may be suspected if a bruit is present but, in essence, those lesions must be diagnosed by arteriography.

7. Occlusive lesions of the great vessels at their origin from the aorta are best managed with bypass grafts. Similar thrombosis of an internal carotid or vertebral artery is usually corrected by endarterectomy and only occasionally with a bypass graft.

8. Local anesthesia is used whenever possible to permit immediate detection of neurologic changes during operation and to avoid certain hypotensive effects which often accompany general anesthesia. Even brief drops in blood pressure may result in hemiplegia.

9. The results of surgical correction of extracranial arterial thromboses producing brain ischemia are excellent in suitable cases.

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CHAPTER 3

Diseases Involving the Arterial Supply to the Upper Extremities

DESPITE the spectacular advances that have been made in the management of many arterial diseases in recent years those involving the upper extremity have received relatively little attention. It is true that these vascular lesions do not often cause massive gangrene since the collateral circulation of the arm is extensive. Nevertheless the symptoms which result from relative ischemia can be most annoying and even incapacitating. And in the occasional patient partial amputation of the extremity will be required if occlusion of the subclavian axillary or brachial artery cannot be relieved.

Most diseases which affect arteries elsewhere in the body may occur in the upper extremity but certain conditions are particularly apt to involve the vessels of the arm and some are peculiar to this member. The following will be considered:

A Thoracic Outlet Syndromes

1. Scalenus Anticus Syndrome
2. Cervical Rib Syndrome

B Occlusive Disease of Major Arteries

C. Reflex Neurovascular Dystrophy

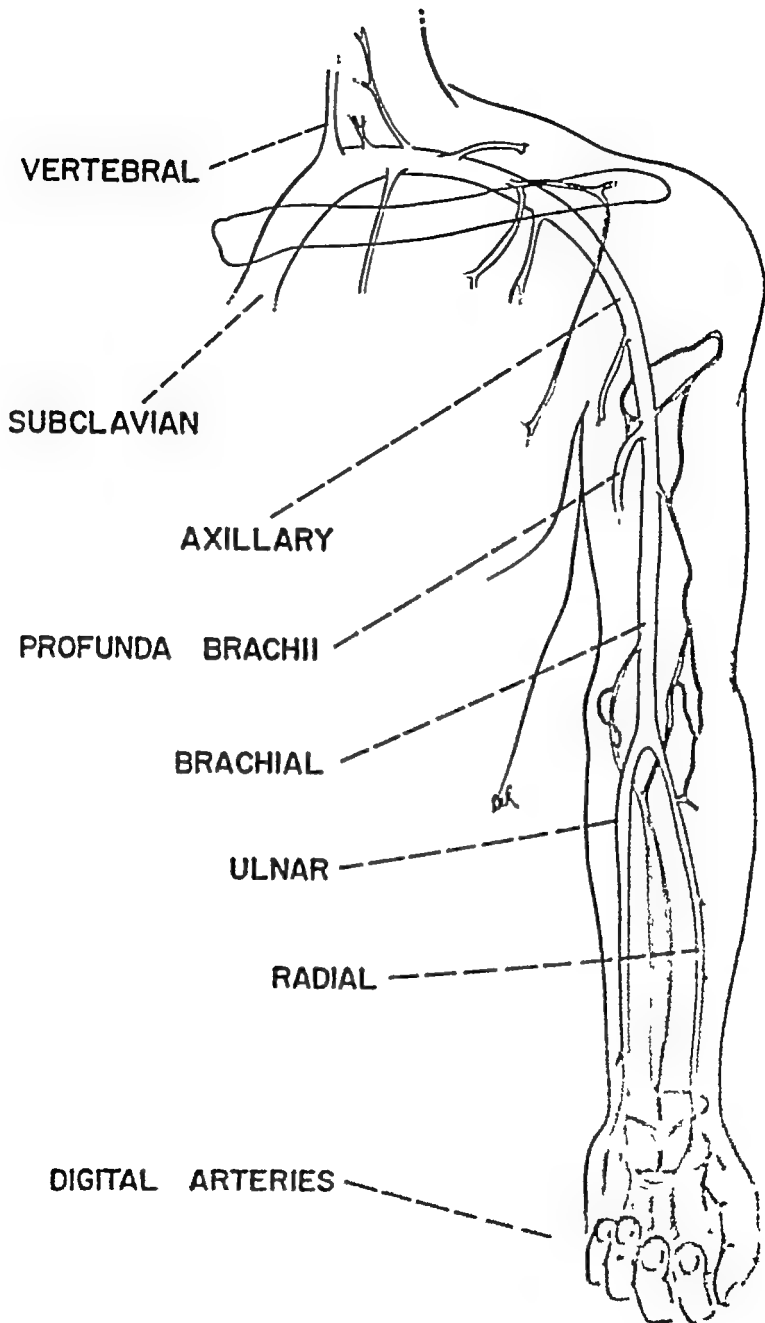


FIG. 16 The collateral arterial supply to the upper extremity is extensive, and major gangrene following arterial

- D Raynaud's and Other Vasospastic Diseases
 - 1 Raynaud's Disease
 - 2 Vibrating Tool or Percussion Syndromes
 - 3 Syndrome of the Middle Aged Female
 - 4 Scleroderma
- E Buerger's Disease (Thromboangitis Obliterans)
- F Obliterative Atherosclerosis of the Digital Arteries
- G Aneurysms Embolism and Traumatic Injuries

CENTRAL CONSIDERATIONS

Anatomy The arterial supply to the upper extremity is shown in Figure 16. The left subclavian artery arises from the aortic arch and the right subclavian artery arises as a branch of the innominate artery. These vessels may be occluded at their origins or at any point along their course and the incision used for exposure of the occluded subclavian artery will depend upon the segment that is diseased. The intra-thoracic portion is best approached using a sternal splitting incision and the supraclavicular portion is exposed through an incision above and parallel to the clavicle. The axillary artery may be visualized using an incision in the delto-pectoral groove with or without division of the tendon of the pectoralis major muscle as it inserts on the humerus. It is not usually necessary to divide this tendon for one can work above and below it. The rest of the arteries of the arm are

occlusion is far less likely to occur in the arm than in the leg. Nevertheless, even if gangrene should not develop a relative ischemia can result in considerable disability. Thus an effort should be made to restore normal arterial flow wherever possible. Severed arteries at virtually all levels, including those at the wrist have been successfully anastomosed.

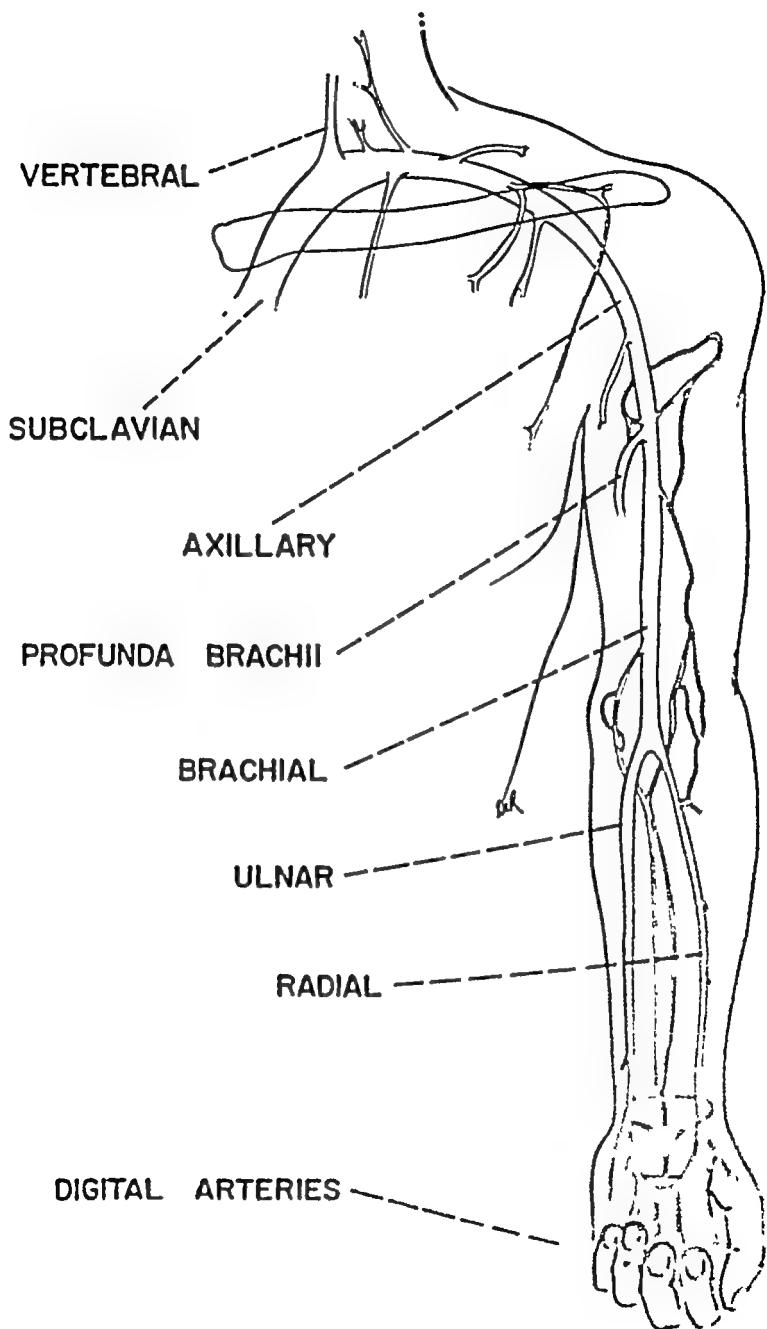


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- 4 Scleroderma

E Buerger's Disease (Thromboangitis Obliterans)**F Obliterative Atherosclerosis of the Digital Arteries****G Aneurysms, Embolism and Traumatic Injuries****GENERAL CONSIDERATIONS**

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and these features have been most commonly described under either the scalenus anticus syndrome or the cervical rib syndrome, or a combination of the two. These two conditions may overlap considerably, and a taut scalenus anticus muscle compressing the subclavian artery may also be associated with a cervical rib.

Scalenus Anticus Syndrome Without Cervical Rib

Anatomy and Pathophysiology. The structures which participate in the scalenus anticus syndrome^{14-20, 21} are shown in Figure 18. The subclavian artery is compressed as it emerges from the thorax posterior to the scalenus anticus muscle and the first thoracic rib. The scalenus anticus syndrome is often found in women with low sloping shoulders, and it can be aggravated in some patients by deep inspirations. The scalenus medius muscle has been incriminated in some subjects. In fact, in one patient we operated upon there was a firm fibrous band running along the anterior border of the scalenus medius muscle, and this band appeared to be causing at least a part of the indentation of the subclavian artery.

There is no general agreement regarding the manner in which the scalenus anticus muscle produces subclavian artery compression. Among the deviations reported by different surgeons have been an unusually broad insertion of the scalenus anticus along the first rib, fibrosis of this muscle, and hypertrophy with abnormal spasm. Nevertheless, no particular pathology has been consistently observed in the scalenus anticus muscle, and it would appear that a combination of

factors results in the compression of the subclavian artery. In addition to actual compression of the vessel itself there may also be a type of Raynaud's phenomenon due to reflex vasospasm from the arterial compression or from activation of elements of the brachial plexus.

Clinical Findings The patient most often a woman usually consults the physician because of pain coldness cyanosis tingling paresthesia fatigue or numbness in the arm and/or hand when it is elevated above the head (Fig. 19) and at times even when

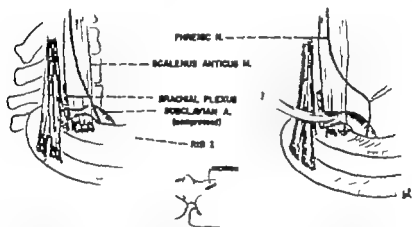


FIG. 18 The elements of the brachial plexus and the subclavian artery emerge between the scalenus anticus and scalenus medius muscles to supply the arm. In some patients, in a manner that is not always clearly perceptible the scalenus anticus muscle compresses the subclavian artery. This is often aggravated by turning the head to the side of the lesion or by elevation and hyperabduction of the arm. Atherosclerotic plaques may form at the site of compression and repeated trauma. Many patients can be managed conservatively but the more severe cases may require division of the scalenus anticus muscle with or without cervical sympathectomy.

it is at the side. Pain may also be felt in the supraventricular area and up the side of the neck. When symptoms occur intermittently with the arm at the side and the radial pulse present, an irritative vasospastic element should be suspected. However, when the arm is elevated above the head and abducted the there may be enough compression of the subclavian artery

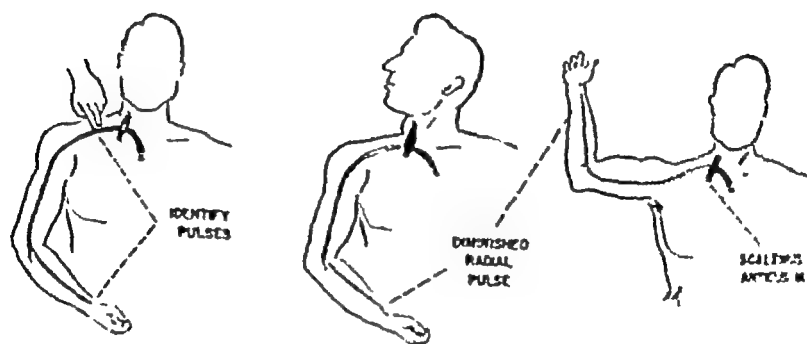


FIG. 19 Adson³ described several important aspects of the scalenus anticus syndrome, including obliteration of the radial pulse by turning the head to the involved side. Hyperabduction of the arm and turning the head sharply to the opposite side may also diminish or obliterate the pulse.

to produce ischemia of the fingers due to obliteration of pulsatile flow. The patient may complain of soreness or numbness, or that the arm "goes to sleep" when she is performing work which requires elevation of the arms above the head, such as that of hanging clothes on a clothes line, the involved arm may tire very quickly and have to be lowered again.

Obliteration of the pulses may also be produced by leaving the arm at the side and turning the head sharply towards the direction of the affected extremity. The same effect of compression is produced in

some patients by turning the head sharply to the side opposite that in which the symptoms have occurred. In addition to changes in the pulse and blood pressure one may feel a thrill in the suprascapular space on the involved side and a bruit may be heard at this point. It has been noted by us and others that the bruit and other findings indicating compression of the subclavian artery may be exaggerated by asking the patient to take a deep breath. If the compression of the subclavian artery has resulted in dilatation or "aneurysm" formation of the subclavian artery distal to the scalenus anticus a pulsating mass may be felt. Atherosclerotic plaques may form even in the absence of dilatation. If the blood pressure in both arms is normal and the values are essentially equal with the arms at the sides when the patient is supine occlusion by thrombus or by embolus is essentially excluded. Again the scalenus anticus syndrome may be associated with a cervical rib but at this point in the discussion we are confining our remarks to the scalenus anticus syndrome where there is no cervical rib.

One can be misled regarding the clinical significance of the diminution in the volume of the pulse when the various maneuvers described above are performed for the pain may be due to an entirely different cause. In other words many normal subjects who have no symptoms whatever may have a positive Adson test indicating compression of the subclavian artery and several other conditions which must be considered in the differential diagnosis of scalenus anticus pain are listed in Table I. For example, we recently saw a patient who was being considered for surgical correction of a scalenus anticus syndrome when it became clear

that his symptoms were due to compression of the spinal nerves by marked cervical osteoarthritis.

Arteriograms are not usually necessary.

Management. In many patients conservative measures consisting of reassurance, sedation, and physiotherapy suffice to relieve the symptoms, and gradually the condition may subside. In others, however, the reduced blood flow produced when the arm is elevated above the head or hyperabducted during sleep is such as to constitute a genuine indication for surgical intervention. At operation under general anesthesia the

TABLE 1 *Some Causes of Pain in the Shoulder, Arm, or Hand*

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- | | |
|---|--|
| 1 | Nerve Lesions |
| a | Cervical disc (herniated) |
| b | Spinal cord tumor |
| c | Superior sulcus tumor |
| d | Neoplasm of brachial plexus (as in neurofibromatosis) |
| e | Peripheral neuritis |
| f | Traumatic injury to brachial plexus |
| 2 | Vascular Lesions |
| a | Scalenus anticus syndrome |
| b | Cervical rib syndrome |
| c | Arterial thrombosis |
| d | Aneurysm |
| e | Raynaud's disease |
| f | Thromboangitis obliterans |
| g | Vibrating tool (percussion) syndrome |
| h | Reflex neurovascular dystrophy |
| i | Obstructive atherosclerotic disease of terminal arteries |
| 3 | Miscellaneous |
| a | Cervical osteoarthritis |
| b | Bursitis |
| c | Referred pain from the diaphragm, heart or below |
| | trunk |

supraclavicular space is best explored through a transverse incision above the clavicle (Fig 18). The skin platysma muscle, omohyoid muscle and the clavicular head of the sternocleidomastoid muscle are divided. This allows one to identify the scalenus anticus muscle along the anterior surface of which runs the phrenic nerve which must be identified and retracted out of harm's way. Next the location of the subclavian artery is identified by palpation and the elements of the brachial plexus running immediately adjacent to this vessel but superior to it are exposed and gently retracted. Once the point of exit of the subclavian artery between the scalenus anticus and the scalenus medius muscles has been identified one can determine the effect of turning of the head towards the side of the lesion on the subclavian pulsations. In one patient we found that the scalenus anticus muscle very definitely did compress the left subclavian artery when the head was rotated to the involved side by the anesthesiologist. Here the effect of the scalenus anticus muscle was augmented by the fact that a hard fibrous band ran along the anterior border of the scalenus medius muscle giving a firm structure against which compression by the scalenus anticus muscle could be more effective. Once the important structures of the area have been carefully exposed and the nerves gently retracted the scalenus anticus muscle is divided near its point of insertion on the first rib. This usually serves to free adequately the subclavian artery. The thoracic duct enters the angle made by the junction of the left subclavian and internal jugular veins and care is required to avoid injury to it as the scalenus anticus is divided near its insertion.

Sympathectomy is occasionally indicated as an additional measure following division of the anterior scalene muscle, though it is more often performed as a part of the surgical treatment of the cervical rib syn-

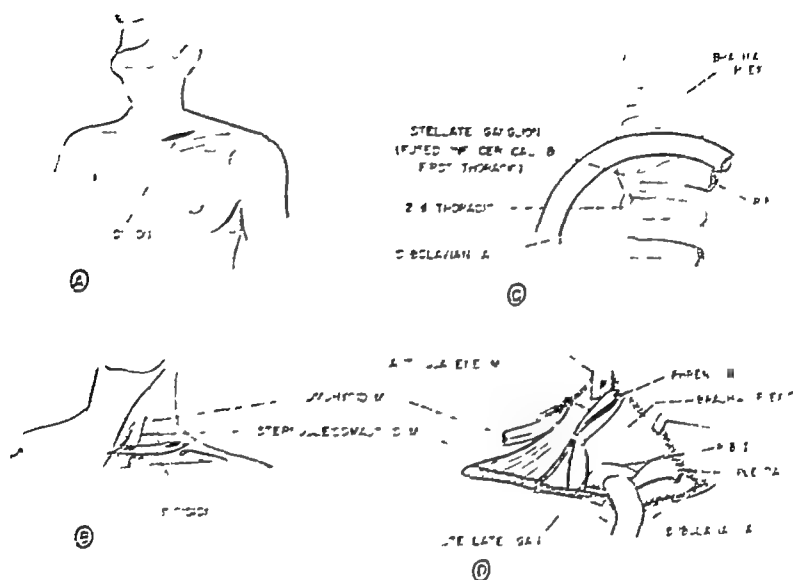


FIG 20 An adequate cervical sympathectomy, with excision of the stellate ganglion, results in a Horner's syndrome. The incision shown (A) is deepened to include division of the clavicular head of the sternocleidomastoid muscle and the omohyoid muscle (B). The subclavian artery (C) is identified by palpation and is then dissected free from the brachial plexus above it. The vessel is then retracted downward and forward to expose the pleura and the first rib posteriorly just below the thoracic inlet. The pleura is then elevated from the first two ribs using blunt dissection. The inferior cervical ganglion (or stellate ganglion if the inferior cervical and first thoracic are fused) can then be palpated as a subfascial structure about the size and shape of a shelled peanut lying against the neck of the first rib (D). We prefer to excise the stellate ganglion, the second thoracic ganglion, and the chain between them.

drome. We prefer the supraclavicular approach to the stellate ganglion (Fig. 20). The subclavian artery is retracted downward and forward and the fascia is divided to permit blunt dissection of the pleura from the first and second ribs posteriorly. The inferior cervical ganglion (which when fused with the first thoracic ganglion below is termed the stellate ganglion) is a rather large structure being about the size of a shelled peanut. It lies on the neck of the first rib. The sympathetic chain may be found and palpated beneath the endothoracic fascia. If at times the inferior cervical ganglion is difficult to locate the sympathetic chain can be identified along the necks of the ribs posteriorly and traced upward. Most surgeons agree that in performing an adequate cervical sympathectomy the inferior cervical ganglion (or fused stellate) must be excised and a Horner's syndrome accepted.

Yet sympathectomy is not commonly required. As a rule division of the scalenus anticus muscle along with any firm fibrous band which may be running in front or behind the subclavian artery is sufficient to free the vessel from compression in our experience. Rarely thrombosis of the artery may exist due to repeated trauma at the site of compression and post stenotic dilatation may develop. Such dilatation of the vessel is however more common in association with the cervical rib syndrome than with the pure scalenus anticus syndrome. Postoperatively the blood pressures in the two arms should be normal and equal regardless of posture or position of the extremity.

Lastly it is to be emphasized that the patient in whom an operation is required for a genuine scalenus anticus syndrome is relatively rare.

Cervical Rib Syndrome

Anatomy²⁹ and Pathophysiology In contrast to the scalenus anticus syndrome, in which the precise cause of the arterial compression is at times difficult to demonstrate clearly, the cervical rib syndrome is associated with far more definite anatomic derangements. In Figure 21 are shown the usual findings when a cervical rib is present and is compressing the subclavian artery. The artery is most likely to be encroached upon when the cervical rib is complete and when the distal end of this structure is enlarged where the artery passes over it. Of course, the cervical rib may vary in length from a very short stubby structure—or a sharp pointed one with a long fibrous attachment to the first rib—to a complete rib. Ross⁴⁸ has pointed out that if the cervical rib is a complete one, articulating with the first rib, the brachial plexus often comes off one segment higher up and neural compression is not a prominent feature. In this circumstance, however, the subclavian artery may be compressed as it crosses over an enlargement of the distal end of the cervical rib (Fig 21). (Some authors have portrayed the anatomic situation with the subclavian artery passing between the cervical rib and the first rib but, in our experience, the artery has passed above the cervical rib.) In contrast, when an incomplete cervical rib is short and pointed, a fibrous band may connect the distal end of the rib to the first thoracic rib, in this circumstance compression of the nerve roots³⁶ may result in a clinical picture of segmental sensory loss with wasting of the small muscles⁴² of the hand, but there is less likelihood that the subclavian artery will be encroached

upon. Thus in one circumstance one may have largely vascular manifestations of the cervical rib and in another largely neurologic manifestations.

Although the cervical rib is present at birth further development in its size and its ability to compress the subclavian artery occurs with growth. In fact relatively few patients develop symptoms prior to the age of 20 though one girl we treated was only 15 years of age.

Of course, many persons have unilateral or bilateral cervical ribs which never produce serious symptoms. Nevertheless patients with a symptomatic cervical rib should be followed carefully. Shienkin²¹ reported three patients with a cervical rib who had sustained subclavian artery thrombosis all of whom had had

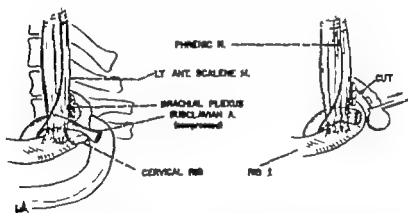


FIG. 21 The cervical rib may be seen compressing the subclavian artery as it courses over the enlarged bony tip. Arm pain may be due to both ischemia and/or pressure upon the brachial plexus. Thrombosis of the artery at the point of compression is not rare. Treatment consists of excision of the distal two-thirds of the rib with or without division of the anterior scalene muscle and at times cervical sympathectomy.

mild symptoms for several years preceding this serious complication. In fact, the diagnosis of cervical rib syndrome had been made early in each instance, but conservative management had been advised.

The *vascular complications* ^{1, 15, 30, 40, 48, 50} that may develop secondary to cervical rib compression are more often serious than those which are likely to follow scalenus anticus syndrome without cervical rib. First, there may be marked pain due to reduced blood flow in the involved extremity. In one patient that we treated the subclavian artery was so small that in the early stages of the dissection it was thought to be some small aberrant vessel, and it was preserved only with indifference. Once the cervical rib had been excised, however, the small vessel proved to be the subclavian artery itself. Second, there may be post-stenotic dilatation or aneurysm formation ³⁵. Third, thrombus may form in this aneurysm and pieces of it break off to embolize to the more distal arteries of the extremity. Fourth, occasionally a thin-walled aneurysm may rupture. Fifth, thrombosis ^{8, 30, 45, 40, 51} may occur at the point of compression in the absence of an aneurysm, and acute occlusion can cause gangrene in the arm or hand.

Clinical Findings The symptoms, which include various signs of nerve compression or ischemia of the extremity, have been alluded to previously. These consist of numbness, tingling and paresthesia of the arm and hand, at times associated with pain in the supraclavicular area and the same side of the neck or of finger pallor, cyanosis, or even sites of ulceration along the lateral border of the nails or at the finger tips, or of intermittent claudication in the arm or

atrophy of the intrinsic muscles of the hand perhaps associated with trophic changes in the fingers and nails. One should not be misled by intermittent attacks of vasospastic phenomena suggestive of Raynaud's disease these being due to irritation of the elements of the brachial plexus or the subclavian artery with resulting reflex vasospasm. The possibility that such findings are the result of a scalenus anticus cervical rib or atherosclerotic occlusive process should always be considered in the differential diagnosis.

Once the symptoms have suggested the possibility of cervical rib syndrome the determination of the blood pressure in the two arms particularly with the arm on the affected side being placed in different positions to detect compression of the subclavian artery is helpful. A supraclavicular lump may represent the enlarged end of a cervical rib and an unusually prominent subclavian artery may reflect anterior displacement of this vessel by the anomalous rib. Occasionally one will note a thrill and a bruit in the supraclavicular fossa. In addition to a diminished blood pressure and pulse on the affected side the possibility of a cervical rib syndrome is further enhanced by the finding of a cervical rib on chest roentgenogram and occlusion of the subclavian artery can also be demonstrated by arteriogram. A sudden increase in the severity of the symptoms suggests sudden thrombosis or extension of a previous thrombus.

Management The mere presence of a cervical rib, perhaps noted on routine chest x ray does not constitute an indication for surgical intervention. And even when symptoms are present most surgeons have

previously felt that one might with justification elect either conservative management with physical therapeutic measures or surgical intervention. Recently, however, in part because of the reports of thrombosis and serious disability resulting from previously symptomatic cervical ribs,^{8, 48, 49, 51} the writer has advised earlier excision of symptomatic cervical ribs—with or without scalenotomy³ and cervical sympathectomy as indicated. In other words, we feel that it is important to perform prophylactic surgery before thrombosis of the subclavian artery and distal embolization have occurred. Thromboendarterectomy of extensively involved arteries is often unsuccessful, and the relatively few direct grafts and bypass grafts that have been used in the upper extremity have exhibited a rather discouraging incidence of early thrombosis.

At surgery the exposure shown in Figure 18 is used to excise the anterior two-thirds of the offending cervical rib (Fig 21). Care is exercised to avoid injury to the artery as it crosses over the distal end of the cervical rib, for at this point the wall of the vessel may be considerably thinned. The phrenic nerve should be retracted out of harm's way, and special gentleness is employed in retracting the elements of the brachial plexus to afford sufficient exposure to permit excision of the appropriate amount of the cervical rib. It is very easy to retract the brachial plexus with a firmness which will result in considerable pain in the arm and hand for some time postoperatively. In fact, such postoperative distress along the medial surface of the arm and the ulnar portion of the hand can be more incapacitating than was the cervical rib syndrome itself.

Once the cervical rib has been excised the subclavian artery may be seen to dilate even before the wound is closed. However it will dilate even further in subsequent days. Following freeing of the artery from compression by excision of the rib it is important to determine whether or not thrombosis of the artery exists. If thrombosis with occlusion of the artery has developed one must decide whether thromboendarterectomy is feasible (it often is if the process is not extensive) or whether a bypass graft should be used or whether only cervical sympathectomy is feasible. Extensive subclavian artery thrombosis has heretofore been treated largely by cervical sympathectomy but at the present time a more aggressive approach aimed at restoration of pulsatile flow is being generally adopted. Unfortunately in some instances extensive distal embolization will have occurred.¹⁰

OCCCLUSIVE DISEASE OF MAJOR ARTERIES

Atherosclerotic occlusive disease may affect any artery in the body and the arteries of the arm are no exception. We have treated patients with thromboses of the subclavian artery at its origin from the aorta of the subclavian artery above the clavicle the axillary artery and the brachial artery. Actually once a thrombus has formed at a given point it tends to propagate distally and occasionally proximally. Thus it is of great therapeutic importance to make the diagnosis of thrombosis early in the course of the disease, so that successful thromboendarterectomy can be performed. Unfortunately many of these patients are accurately diagnosed only after the thrombosis in say the axillary or subclavian artery has propagated for a

considerable distance distally. Under such circumstances it may not be possible completely to restore pulsatile blood flow through the artery. For whereas a short segment of artery can be endarterectomized quite satisfactorily, a long segment can be endarterectomized only with a considerable hazard of fresh clot formation postoperatively.

Clinical Findings The signs and symptoms found in the patient with occlusive disease of a major artery of the arm range all the way from the very mild to the most severe with actual gangrene which requires amputation. The first symptom which the patient notices may be a relative coldness of the hand on the involved side, perhaps associated with pain on the exercise of this extremity or upon exposure of this extremity to cold. A surprisingly extensive degree of occlusion of a major artery of the arm may be well tolerated without significant symptoms in the absence of exercise. However, if the patient attempts to use this arm for vigorous effort, intermittent claudication may develop. Another frequent finding in the presence of major occlusive disease is secondary Raynaud's phenomenon on exposure of the hand to cold (Fig. 22). Here even the normal degree of vasospasm may result in marked digital ischemia. And, of course, marked and prolonged ischemia will produce trophic changes such as deformed nails, thinning of the pulp of the fingers with shiny tightening of the skin, and upon occasion indolent ulcers. In contrast to the findings which one will note when the digital arteries to a single finger have been occluded, where the other fingers may have a normal temperature, thrombosis of a major artery may cause the entire hand to be cooler

than its opposite member though even then one finger may be affected more than the others and this is reflected in initial ulceration in a single digit

Physical examination will also show in addition to the various signs of ischemia just noted a diminished blood pressure below the level of arterial occlusion. Thus if the radial pulse is present and of good volume one might suspect that the arterial pathology is distal to this point. And usually this would be a cor

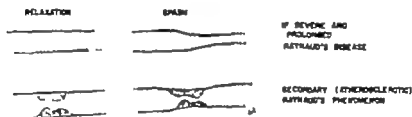


FIG 22 Raynaud's disease at least in its early stages consists of pure spasm of the smaller arterial structures (above) Raynaud's phenomenon in contrast, may also be caused by even normal degrees of digital spasm on exposure to cold when partial organic vascular occlusion already exists (below)

rect assumption though it is not always possible to detect partial proximal occlusion of a major vessel merely by palpation of the pulse. The collateral circulation may be so extensive that partial occlusion of the main artery will be evident only when a blood pressure cuff an oscillometer or a plethysmograph is employed to demonstrate differences in the volume of blood flow between the two upper extremities. For example ligation of the subclavian artery in various operative procedures (e.g. Blalock anastomosis) may abolish the radial pulse for only a few hours. Nevertheless a weak or absent radial pulse should

prompt examination of the brachial, axillary, and supraclavicular subclavian pulses. On the right, absence of the subclavian pulse but presence of the common carotid pulse identifies the level of occlusion as being in the first portion of the former. However, if both the carotid and subclavian pulses are absent, the occlusion is in the innominate and a bypass graft from the ascending aorta may be indicated. In this manner one can establish the approximate level of occlusion. It is surprising how often patients with major arterial occlusive disease are still diagnosed as having "Buerger's disease" (thromboangitis obliterans) when the radial or even the brachial pulse is absent. When the major pulsations at the wrist and above the elbow are absent, the diagnosis of thromboangitis obliterans or of Raynaud's disease is clearly untenable. We treated one patient who had had a cervical sympathectomy for suspected Buerger's disease, when he had complete occlusion of the subclavian artery. Unfortunately, this occlusive disease had resulted in a propagating thrombus which extended all the way down to the bifurcation of the brachial artery, a normal blood flow could not be restored to the extremity, and eventually the arm had to be amputated just above the elbow. In another patient, however, who had also been diagnosed as having Buerger's disease, a thrombus in the axillary artery was easily removed and an excellent postoperative radial pulse was associated with eventual clearing of the gangrene which had developed at the tip of one finger.

Once the probable presence of arterial occlusion has been established by palpation of the artery at various levels and the observation of other clinical findings

just mentioned the use of an arteriogram may be most helpful in precise localization of the site and extent of occlusion. Since from the level of the scalenus anticus distally the arteries are readily available for arteriographic visualization it is usually a simple matter to perform this study.

Management. Every effort should be made in most patients to restore normal or pulsatile blood flow. We have thus far depended entirely upon thromboendarterectomy (Fig. 23) in the restoration of adequate

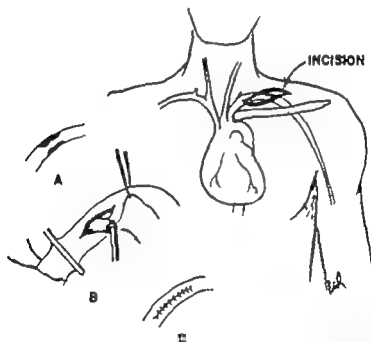


FIG. 23. Atherosclerotic thrombosis may occur at any level of the arterial tree in the arm (or in the entire body). If treated early before extensive propagation of the thrombus has occurred thromboendarterectomy can be performed successfully for lesions of the larger arteries in most instances. The use of grafts in the arm has resulted in thrombosis in a considerable number of cases.

blood flow through the arteries of the arm. Although we would not hesitate to employ a bypass graft where indicated, those who have used grafts have reported a considerable incidence of early thrombosis.

In the event that a completely successful thromboendarterectomy cannot be performed, perhaps because of the extent of the disease, a cervical sympathectomy on the involved side is advisable. It would seem prudent also to perform a sympathectomy along with the use of a graft, to assure maximal blood flow should the graft thrombose. Again, an adequate cervical sympathectomy will usually result in a Horner's syndrome on the involved side. Exposure of the arteries of the arm is exceptionally easy, compared with most arteries elsewhere in the body, for a short incision directly over the involved vessel usually is all that is required.

REFLEX NEUROVASCULAR DYSTROPHY

The as yet poorly understood process termed reflex neurovascular dystrophy^{13, 17} may be caused by a variety of disease processes. Such unrelated conditions as traumatic neurovascular injury, cold injury,¹⁸ myocardial infarction, and cerebrovascular hemorrhage may all result in this picture of sympathetic nervous system dysfunction, not dissimilar to the causalgia of the lower extremity which often follows neural or vascular injury which need not divide major structures. There is often marked vasospasm and intermittent episodes of secondary Raynaud's phenomenon. The symptoms which these patients experience thus include vasomotor disturbances with coldness, excessive sweating, and edema of the arm and particularly the

hand. If the condition persists there may develop atrophy, hyperesthesia and paresthesia. Often the pain is of a severe and burning type with trigger points, the stimulation of which may exaggerate the condition. There may be extensive demineralization of the bones of the arm and hand far in excess of what one might expect from disuse alone (Sudeck's osteoporosis⁸⁷).

Sympathetic nervous system dysfunction is the common denominator which can be detected in the variety of clinical conditions which may cause reflex neurovascular dystrophy. Most of the findings that are characteristic of the condition can be caused by such dysfunction and the process is often relieved considerably by sympathectomy when this is absolutely necessary. Again the shoulder-arm-hand syndrome⁴² is similar in many respects to the typical causalgia which may affect the lower extremity following a gunshot wound in which neither a major nerve nor an artery has been severed. This pain in the lower extremity has almost by definition to be relieved by sympathetic nerve block or sympathectomy to merit the term "causalgia."^{11, 42} And similarly relief of causalgic pain in the upper extremity is often obtained by cervical sympathetic nerve block. The reason that cervical sympathectomy has not been performed more often for the shoulder-hand syndrome with its causalgia-like components in the upper extremity as compared with the frequency of sympathectomy for causalgia in the lower extremity is the fact that Horner's syndrome usually follows an extensive cervical sympathectomy in addition to the fact that the operative exposure is more involved than that required for lumbar sympathectomy.

Management If one can find definite etiologic entities, such as occlusive disease or a cervical rib, then specific therapy will be directed towards the offending pathology. However, in many instances no definite organic lesion can be demonstrated to account for the various signs and symptoms which constitute the shoulder-hand syndrome of neurovascular dystrophy, and here one must employ less specific measures. Sedation, reassurance, and aggressive physical therapy can do much to prevent the development of chronic shoulder-hand syndrome. At times stellate ganglion block may produce considerable temporary relief and, if so, cervical sympathectomy should be considered in the more severe and protracted cases. Nevertheless, in our own experience it has usually been possible to avoid a formal cervical sympathectomy. In passing, it may be noted again that all elements of neurovascular dystrophy or shoulder-hand syndrome may follow forceful retraction of elements of the brachial plexus in the course of exposure of various structures for operations in the supraclavicular space.

RAYNAUD'S AND OTHER VASOSPASTIC DISEASES

Vasospastic diseases^{1, 37} of the upper extremity still constitute a rather complex and poorly understood group of lesions. Even so, certain definite advances in our knowledge of these various conditions have been made. Although vasospasm superimposed upon organic lesions may give rise to the typical cyanosis and other evidences of digital ischemia, the so-called secondary Raynaud's phenomenon, there doubtless

are conditions which are largely spastic in nature at least in their initial stages (Fig. 22). Among these are early states of Raynaud's disease, vibrating foot or periorbital syndromes, the syndrome of the middle-aged female and scleroderma.²⁰ However, it should be emphasized that even in these conditions which initially may be largely vasospastic in nature there eventually may develop arteriolar thickening and thus a degree of organic occlusion of the involved vessels. Once luminal narrowing has occurred due to thickening of the arterial wall, it takes much less vasospasm to produce a severe ischemia of the digits than was the case initially when the lumina of the arteries and arterioles were normal between attacks.

Raynaud's Disease

In 1862 Raynaud²¹ described a condition of local asphyxia and symmetrical gangrene of the upper extremities which thereafter came to bear his name. Unquestionably a number of the cases which he included in his report were patients who had varying degrees of organic occlusion with superimposed vasospasm (Raynaud's phenomenon rather than Raynaud's disease²⁷). Nevertheless there are patients who initially at least suffer from a purely vasospastic lesion. The patient is usually a female and both hands are affected. Lewis and his associates²²⁻²⁴ demonstrated that a major change in the vessels of the digits of the patients with Raynaud's disease—the so-called local fault—is an increased sensitivity of these vessels to cold as compared with the sensitivity of normal persons. A lesser degree of vasospasm may also be precipitated in some women by emotional crises.

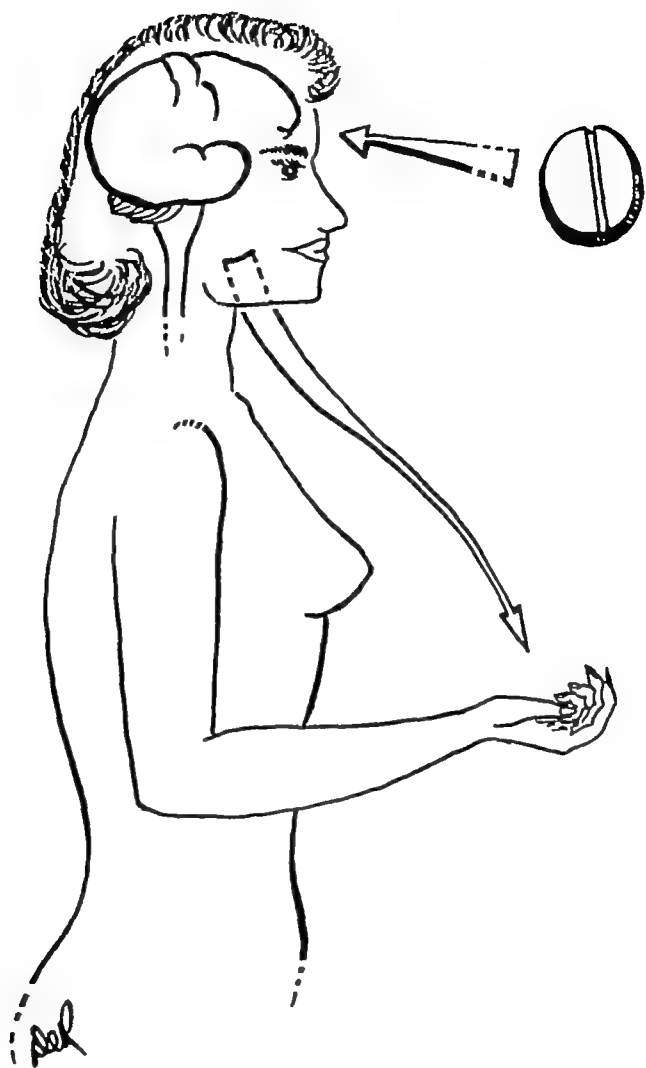


FIG 24 It has been found that the patient having an attack of Raynaud's disease may simultaneously exhibit electroencephalographic changes, indicating a central origin of the vasomotor nerve impulses in some instances. The successful control of mild cases with tranquillizing drugs has been reported ¹²

and it has been found that the more mild cases of this condition may be controlled with tranquilizing drugs (Fig 24). This local fault—consisting of an increased sensitivity of the digital arteries and arterioles to cold—persists in some degree even after a cervical sympathectomy has been performed.²¹

Management. By careful conservative treatment cervical sympathectomy can be avoided in most instances. The patient should avoid exposing either the body or the hands to chilling. Thus warm clothing and warm gloves must be worn. De Lorkats¹² has pointed out that electroencephalographic changes have been found in patients during attacks of Raynaud's disease and it was noted above that mild cases have been managed successfully with tranquilizing drugs. Priscoline given orally in doses of 25–50 milligrams three or four times daily has also been helpful in our experience. If 50 milligrams every six hours produces the disagreeable chilly sensations which many patients find annoying the dosage should be temporarily reduced to 25 milligrams four times a day; later it is often possible to increase the dosage again to 50 milligrams without producing the undesirable side effects.

If conservative measures do not suffice the patient may eventually require cervical sympathectomy.² Most surgeons agree that the best and most lasting results are achieved by an extensive sympathectomy which removes the inferior cervical ganglion and the first two thoracic ganglia below it. Thus the presence of a Horner's syndrome is virtually essential to demonstrate adequate sympathectomy. Of course this disfigurement is less objectionable if sympathectomy

must be performed bilaterally with resulting symmetry even though bilateral lid droop exists

In certain patients the beneficial effects and relief provided by cervical sympathectomy are short-lived. This has been explained by some as being due to the further development of alternate sympathetic pathways to the digital vessels. Such pathways may pass through the spinal nerves and thus not be amenable to excision in the course of sympathectomy. Others have felt that increased local sensitivity of the digital vessels to cold or to pressor amines might be responsible. And of course incomplete sympathectomy or nerve regeneration is at times a possibility.

Vibrating Tool or Percussion Syndromes

In 1918 Hamilton²³ described the effect of the anvil hammer on the hands of stonecutters. Since then it has become increasingly apparent that various types of occupations which result in repeated trauma of a percussion type can produce changes which result in an increased sensitivity of the digital vessels to cold.^{4, 5} Actually—and perhaps in a majority of cases—this increased sensitivity to cold reflects not a purely vasospastic phenomenon by a vasospasm superimposed upon luminal narrowing due to intimal thickening and subintimal proliferation. Other vocations which may be associated with such vascular changes are typing, piano playing and situations in which the worker must strike some lever or other object with the palm of the hand or with the sole of the foot. This type of percussion trauma eventually results in changes in the walls of the arteries and arterioles. For example, we have recently seen a patient who had marked vaso

spastic changes in his right foot with cruralgialike pain and trophic changes which apparently were secondary to his occupation in which he was required to strike a lever with the sole of the right foot continuously in operating a press in an industrial plant. A similar change in the hand has been reported.⁴⁷

The common denominator in all these various types of injury would appear to be not the vibration *per se* but the actual striking or percussion force.⁴ As stated although it appears possible that early in the condition there may be a purely vasospastic phenomenon due to an increased sensitivity of the vessels of the hands to cold later there is unquestionably an obliterative fibrosing process which actually diminishes the luminal diameter of the vessels and renders the fingers still more susceptible to ischemia produced by even normal vasospasm of the skin vessels when exposed to cold.

Management Most patients with the percussion syndrome manage to continue work either at the same job or at a different one. In many instances the syndrome is mild and a relatively stoic individual may not even consult a physician. Conservative measures consisting of sedation, vasodilators and protection of the hands from cold are also helpful. At times however as noted in our man who had the changes in his foot secondary to striking the foot pedal of a press the changes are so extensive that ulceration occurs and prolonged disability results. In this particular patient bed rest and local medication had failed to heal a plantar ulcer but lumbar sympathectomy resulted in healing of the ulcer and in a marked increase in exercise tolerance.

Syndrome of the Middle-Aged Female

Representative of the group of vasospastic diseases which may affect the upper extremity but which have as yet been poorly defined is the syndrome of the middle-aged female described by Jepson²⁷. The etiology of this condition is not established, since it occurs too late in life to be primarily inborn and there was in Jepson's cases no evidence of a diffuse atherosclerosis. He found a common feature was that the condition occurred near the menopause, the re-establishment of the menses, either with estrogen therapy or spontaneously, was often associated with improvement. Conservative treatment should be given a prolonged trial before sympathectomy is considered.

Scleroderma

The cause of scleroderma is obscure, but the condition may progress to a point in which it produces gangrene of the tips of most of the digits. It was pointed out by Jonathan Hutchinson²⁴ in 1896 that patients with Raynaud's disease for many years might eventually develop scleroderma in the involved digits. This observation has since been confirmed by others,⁴ and it gives rise to possibly valid speculation that the fibrotic changes characteristic of scleroderma may in fact be the result of fibrosis secondary to long-standing intermittent attacks of ischemia due to arterial and arteriolar spasm. Thus there is always the possibility that the woman with Raynaud's disease may ultimately prove to have or to develop scleroderma. The patients with scleroderma exhibit an increase

in the fibrous tissue and a loss of elastic tissue in the vessel walls with gradual obliteration of the vascular lumen

Management. The treatment of scleroderma is unsatisfactory. The condition may be generalized and the esophageal lesions are particularly striking in some patients. The use of sedation Priscoline and in the carefully selected patient cervical sympathectomy are suggested

BUERGER'S DISEASE (THROMBOANGITIS OBLITERANS)

Just as many conditions formerly called Raynaud's disease actually represented in many instances secondary Raynaud's phenomenon produced by a normal degree of vasospasm in the presence of partial organic occlusion of the smaller arteries of the extremity (Fig 22) so it is with Buerger's disease¹⁸. There can be no doubt that a great many patients previously diagnosed as having thromboangitis obliterans actually had atherosclerotic occlusive disease. Thus we have encountered only one patient in recent years that we thought actually had Buerger's disease—and the diagnosis in even this case was not confirmed by biopsy. As a matter of fact there are those who contend that even the inflammatory and obliterative lesions which occur in the arteries and veins of the extremity in "Buerger's disease" are actually inflammatory lesions secondary to ischemia produced by atherosclerosis¹⁹. Certainly we have treated a number of patients who had been diagnosed previously as having "Buerger's disease" but who actually had atherosclerotic occlusion of the subclavian axillary brachial or more dis-

tal arteries. Therefore one should be skeptical of any diagnosis of thromboangitis obliterans lest atherosclerotic occlusive disease be the etiologic agent.

The principal pathophysiology of thromboangitis obliterans results from the ischemia of the tissues produced by occlusion of the small and at times fairly large arteries such as the radial, ulnar, dorsalis pedis, and posterior tibial. In contrast to Raynaud's disease, which occurs largely in the upper extremities and almost entirely in women, Buerger's disease is more commonly found in the lower extremities and usually but not always in men. The segmental nature of the occlusion in Buerger's disease (and in atherosclerotic occlusive disease) may permit considerable collateral circulation to develop. Superimposed vasospasm (secondary Raynaud's phenomenon) is often a prominent feature of thromboangiitis obliterans.

If a condition such as thromboangitis obliterans does exist, its etiology is largely obscure. The writer has always felt that the condition was related to smoking in many patients, though the disease has been diagnosed many times in patients who did not smoke. The fact that cessation of smoking results in improvement in many patients is of course not diagnostic of Buerger's disease, since the reduced vasospasm would also be beneficial in patients who had atherosclerotic occlusive disease.⁵⁵ It has been pointed out by Linton³⁶ and by Fisher¹⁸ that the widespread use of arteriograms has revealed localized occlusive disease in many patients previously diagnosed as having Buerger's disease. Furthermore, in such patients the availability of biopsy material has established atherosclerotic occlusive disease as the etiologic condition.

rather than the less well defined possibility of thromboangitis obliterans

Finally Thiers⁶ has stated that a process of atherosclerosis occlusive thrombosis and reparative reaction seems to account for the peripheral arterial lesions of thromboangitis obliterans or presenile gangrene. Thus we may be witnessing the disappearance of a disease process as more precise information incriminates a far more understandable and common etiologic agent, namely atherosclerosis.

Management For the moment however let us assume that an occasional patient may be found who will fulfill the more or less poorly defined criteria of Buerger's disease. Such individuals should be cautioned to avoid cold or other trauma to the invalid part. Priscoline therapy may prove helpful and highly important the patient should stop smoking at once if he has been doing so.⁴⁸ Finally sympathectomy has been of much assistance in thromboangitis obliterans as it also has in selected cases of atherosclerotic occlusive disease.

In closing this discussion let it be emphasized again that no patient should be diagnosed as having Buerger's disease until the possibility of atherosclerotic occlusive disease has been excluded insofar as possible by careful physical examination and arteriograms.

OBLITERATIVE ATHEROSCLEROSIS OF THE DIGITAL ARTERIES

Occlusive disease involving the small vessels of the hand and the digits is mentioned only to emphasize that, even though the radial pulse may be present and of good quality atherosclerotic occlusion of the more

distal vessels is not excluded. Thus, even though there is evidence of occlusion of the smaller arteries, neither Raynaud's disease nor Buerger's disease need be incriminated, for atherosclerotic lesions may have resulted in a narrowing of the digital vessels to a point where even normal vasospasm on exposure of the hands to cold results in the secondary Raynaud's phenomenon depicted in Figure 22.

The *treatment* of occlusive disease of the smaller vessels of the fingers is at the present time one of conservatism coupled with sympathectomy in the most severe cases. Resection with grafting or bypassing is not feasible. Protection of the digits from cold or mechanical injury, the use of vasodilator drugs, and sedation for pain—all of these are important. If conservative measures cannot relieve pain and cause regression of indolent ulcers which may develop at the tip of the finger or along the lateral side of the fingernail, cervical sympathectomy should be considered. In contradistinction to Raynaud's disease, where the arteriospasm tends to be symmetrical and bilateral and involves all the digits, though often some far more than others, atherosclerotic occlusive disease of digital arteries tends to be spotty.

ANEURYSMS, EMBOLISM, AND TRAUMATIC INJURIES

Aneurysms Although the general subject of aneurysms will be discussed in Chapter 5, it should be appreciated that aneurysms involve the arteries to the upper extremity not infrequently, though far less often than they do the abdominal aorta or the iliac and femoral vessels. The post-stenotic dilatation or

aneurysm formation which may be associated with a cervical rib has been mentioned. Atherosclerotic aneurysms are found in the subclavian and axillary arteries in the occasional patient. These lesions are managed by excision with restoration of arterial continuity by primary anastomosis or by grafting. Since the incidence of thrombosis in vascular prostheses used in the upper extremity has been fairly common in the relatively few such cases treated it would probably be advisable to perform a cervical sympathectomy by excision of the inferior cervical ganglia and the two adjacent thoracic ganglia at the time of operation since this would diminish the chances of gangrene if abrupt occlusion of the graft should occur. Of course in patients who have had slowly developing atherosclerotic occlusion the collateral circulation is far more extensive than in those with an aneurysm through which blood has continued to flow up until the time of operation.

Embolism. The general problem of embolism is also to be discussed in a subsequent section and it is mentioned here only to emphasize the fact that emboli leaving the heart lodge in the upper extremity in approximately 11 per cent of all cases²². In addition to emboli from the heart there may be extensive distal embolization from simple thrombosis in the subclavian artery beneath a cervical rib or in a post-stenotic dilatation or aneurysm caused by a cervical rib compression. When embolization to the upper extremity occurs prompt intervention and removal of the clot is to be strongly recommended. For whereas actual gangrene of the arm is relatively rare considerable disability due to relative ischemia—re

flected in intermittent claudication and perhaps cyanosis and pain in the fingers—may result from the occlusion of the brachial artery by the embolus. The arteries of the arm are easily exposed throughout almost all of their course, and removal of an embolus, if performed promptly after the episode, should usually prove highly satisfactory. Stellate ganglion block and heparinization may be advisable in the given case to reduce postoperative arterial spasm and to reduce the likelihood of thrombosis at the site of arteriotomy.

Traumatic Injuries The arteries to the upper extremity may be injured in all the ways in which other arteries of the body are injured^{10, 25, 26}. The most common causes of arterial trauma are gunshot, stab, and blunt force wounds. The artery may be lacerated or completely divided, a false or true aneurysm may develop, an arteriovenous fistula may be produced, compression may occur, spasm of the vessel may be severe,^{9, 10} or intraluminal or intimal thrombosis^{31, 43, 46} may produce complete occlusion of the vessel. We have personally treated every one of these injuries in arm vessels. The injury is usually discovered through the history which suggests possible arterial injury, physical examination, and arteriograms. When a serious possibility exists that a major artery has been injured, surgical exploration is often indicated. It has been found that primary suture of divided arteries in the arm is usually successful, and Shaw⁵⁰ has reported successful anastomosis of both the radial and the ulnar arteries.

Further considerations important in the diagnosis and management of arterial injuries will be discussed more fully in Chapter 7.

Summary and Conclusions

1 Arterial diseases of the upper extremity deserve earlier diagnosis and more aggressive management than they have received in the past. For although arterial occlusion in the upper extremity does not often result in large scale gangrene due to the extensive collateral circulation serious and prolonged disability frequently does occur.

2 The following conditions have been reviewed: scalenus anticus syndrome, cervical rib syndrome, major atherosclerotic occlusive disease, reflex neurovascular dystrophy (shoulder-arm-hand syndrome), Raynaud's disease, vibrating tool or percussion syndromes, syndrome of the middle aged female, scleroderma, Buerger's disease, obliterative atherosclerosis of the digital arteries, aneurysms, embolism and traumatic injuries.

3 Anatomic considerations are emphasized and the signs of arterial occlusion at different levels with resulting ischemic changes in the fingers are noted.

4 A digital ulcer or other evidence of finger ischemia may be produced by occlusive disease at the aortic arch, an aneurysm, embolism, subclavian artery compression by a cervical rib, atherosclerotic occlusive disease at more distal levels, thromboangitis obliterans, Raynaud's disease, vibrating tool trauma or scleroderma among others. A common diagnostic error is to conclude that finger ischemia is due to disease in the digit when it is due to an arterial lesion situated far above this level.

5 The scalenus anticus syndrome rarely produces serious vascular complications but compression by a cervical rib not infrequently produces thrombosis and

other pathologic changes. Thus a cervical rib that is producing neural or vascular changes should usually be resected, with or without division of the anterior scalene muscle and sympathectomy as the occasion may dictate.

6 Atherosclerotic occlusive disease may occur at any level, and it unquestionably accounts for the digital ischemia found in many cases of "Buerger's disease." In fact, with improved methods of diagnosis, including biopsy and frequent arteriography, a great many cases of "thromboangitis obliterans" are proving to be instances of atherosclerosis. Thus "Buerger's disease" is becoming rare.

7 Reflex neurovascular dystrophy of the arm reflects a form of sympathetic nervous system dysfunction, and many aspects of the condition resemble the familiar causalgia found in the lower extremity following various types of trauma.

8 The conditions which produce the purely vasospastic Raynaud's disease and those which produce secondary Raynaud's phenomenon are now better understood. It has been found that in some patients with Raynaud's disease the attacks of digital vasospasm are associated with electroencephalographic changes, and mild cases have been successfully controlled with tranquillizing drugs. The secondary Raynaud's phenomenon may be produced by even normal vasospasm in the patient who already has partial organic occlusion of the digital arteries and arterioles.

9 Vibrating tool or percussion trauma may result in an increased tendency to vasospasm and even organic occlusion of small arteries and arterioles in the

involved extremity. The use of an air hammer, piano playing, typing and even striking of the palm of the hand or sole of the foot against levers at work have produced such changes.

10. Virtually any pathologic condition that may affect arteries elsewhere in the body may affect those of the upper extremity.

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CHAPTER 4

Occlusive Disease of the Celiac, Superior Mesenteric, Inferior Mesenteric, Renal, Iliac, Femoral, and More Distal Arteries

GREAT progress has been made in the diagnosis and management of occlusive disease of the abdominal aorta and its major divisions (Fig 25). Following the realization that occlusive disease of the lower aorta and iliac arteries was readily amenable to surgical correction, clinicians began to search for remediable instances of occlusive disease of visceral arteries. It was soon demonstrated that occlusion of the celiac, superior mesenteric, and renal arteries is more common than was formerly believed and that such lesions can be managed by either bypass graft or thromboendarterectomy. It will be the purpose to outline the pathophysiology, diagnosis, and management of occlusive disease involving the visceral arteries within the abdomen, as well as occlusion of the terminal aorta, iliac arteries, femoral arteries and the more distal portions of the arterial bed.

In general, the clinical findings depend upon the location and function of the structures supplied by the arteries in question. Thus thrombosis of the

superior mesenteric artery produces abdominal angina as well as evidence of inadequate intestinal absorption in some patients. Partial occlusion of one or both renal arteries may result in essential hypertension or in late stages renal failure. Occlusion of the terminal aorta results in ischemic pain and at times impotency—the Leriche syndrome. Thrombosis in more distal arteries produces intermittent claudication at appropriate levels and atrophic changes which on cursory examination may be attributed to nerve lesions. Corrective surgery

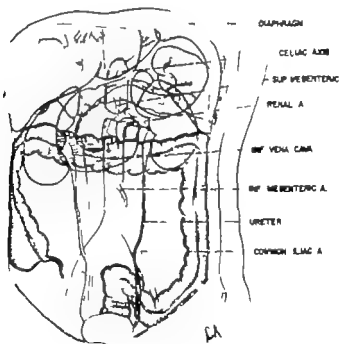


FIG. 25 Occlusive disease of each of the major branches of the abdominal aorta may give rise to more or less characteristic clinical pictures.

is successful in the management of major arterial occlusion in the vast majority of patients

Aortography The diagnosis of partial or complete thrombosis of the branches of the abdominal aorta ultimately depends upon arteriography. A very large number of abdominal aortograms have been performed by one technic or another, but the procedure does entail some risk. McAfee¹⁷ surveyed 13,207 abdominal aortograms reported in reply to questionnaires sent to hospital radiologists and urologists in the United States and found 37 deaths and 98 serious complications. The over-all complications rate of abdominal aortography was 1.02 per cent and the mortality rate was 0.28 per cent. Renal damage from the contrast medium was the most important complication, usually resulting from the injection of excessive amounts of the radiopaque medium, direct renal artery injections, or injections in patients with high aortic obstruction. Neurologic damage was also a significant hazard, sometimes resulting in prolonged morbidity. Less frequent findings included hemorrhage from the puncture site and cardiovascular, gastro-intestinal, and complications of general anesthesia. In reporting their experiences with aortography, Beall and his associates³ emphasized that certain variables were of importance in the incidence of complications and that these included the dosage used, the site of injection, and alterations in hemodynamics in the region of the kidneys. They found that the injection of a renal artery with a large concentration of dye produced severe damage to the kidney. Grossman and Kirtley¹¹ reported paraplegia following translumbar aortography. Skin necrosis occasionally

results from arteriography in the ischemic extremity⁴ and we have seen three such instances ourselves.

Therefore one should employ arteriography only where necessary using no greater volume or concentration of the contrast medium to be injected than is essential. Hypaque is currently preferred.

Arterial Replacements. Numerous reports from different clinics have now appeared in the literature with respect to follow up studies of patients who have had one or another of the various arterial substitutes inserted following resection of an aneurysm or for resection or bypassing occlusive disease. In general aortic homografts²⁰ have been reasonably successful. Nevertheless sporadic reports of dilatation and rupture of such grafts have appeared²¹ and there has been a general tendency to abandon the use of homografts in favor of one of the various artificial materials particularly Teflon^{22, 43, 44} or Dacron^{19, 45}. Formerly Nylon was extensively used but there appears to be fairly wide agreement that complications due to dilatation and rupture are more common following the use of this material than following the use of Teflon or Dacron. Yet, it should be pointed out that less time has elapsed for evaluation of Teflon and Dacron than for evaluation of Nylon. It is possible that with longer follow up studies Teflon and Dacron will prove to be less favorable materials than they are currently considered to be. Arterial pulsations are transmitted through synthetic prostheses.^{19, 21}

The ideal arterial replacement has yet to be developed, but meanwhile those already available represent a tremendous advance in the management of vascular lesions.

OCCLUSION OF THE CELIAC, SUPERIOR MESENTERIC AND INFERIOR MESEN- TERIC ARTERIES

Although mesenteric artery occlusion can be due to an embolus arising in the heart, it has now become increasingly apparent that many instances of mesenteric arterial insufficiency are the result of the same atherosclerotic process which commonly involves arteries throughout the body. Furthermore, as the fairly common syndrome of partial occlusion of the celiac and superior mesenteric arteries by atherosclerotic thrombosis¹⁰³ is becoming more widely known, more and more patients with abdominal symptoms referable to incomplete occlusion of these vessels are being correctly diagnosed and operated upon successfully. Since thrombosis of the celiac artery alone does not often produce dramatic symptoms, celiac occlusion is usually discovered only when the much more serious superior mesenteric artery occlusion has led to aortography and laparotomy.

Celiac Artery

The fact that occlusive disease of the celiac artery is not often diagnosed in the absence of occlusive disease of the superior mesenteric artery is due to the different viscera supplied by the two vessels respectively. The celiac axis supplies chiefly the liver, stomach, and spleen (Fig. 26). It has long been known that accidental ligation of the hepatic artery is associated with a considerable morbidity and a fairly high mortality rate, particularly in the absence of massive antibiotic therapy. Nevertheless, gradual occlusion of the celiac axis could presumably afford

sufficient time for collateral circulation to the organs supplied to develop. For example the stomach might be supplied by the inferior phrenic artery and the inferior pancreaticoduodenal artery among others. The spleen might well become shrunken and fibrotic since it is not essential to life. The liver could similarly obtain some collateral arterial blood supply and it would also receive oxygen and nutrients through the portal vein. Thus whereas acute ligation of the celiac artery may result in serious ischemia of the stomach and liver²² slowly developing occlusion of even the celiac, superior mesenteric, and inferior mesenteric arteries as a group may be tolerated. In 1869 Chiene²³ reported a case in which the origins of the celiac, superior mesenteric, and inferior mesen-

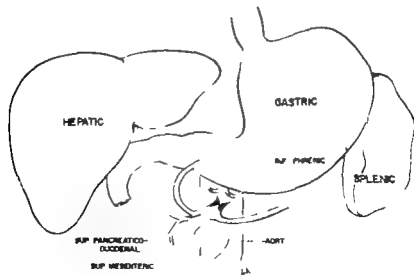
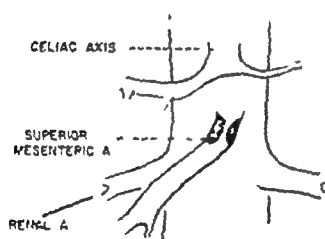


FIG. 26 Thrombosis of the celiac axis is less likely to produce dramatic symptoms than is thrombosis of the superior mesenteric artery

teric arteries and the lower part of the abdominal aorta had all undergone gradual obliteration. The arterial supply of all the viscera within the abdomen was maintained by somatic arteries and by the subperitoneal arterial plexus. These somatic arteries included the left lower intercostal arteries, the lumbar arteries, and the left renal and suprarenal arteries, in addition to the gluteal arteries, the internal pudendal artery, and the right subcostal artery.

It is possible that with more experience cases of celiac artery occlusion, in the absence of superior mesenteric artery occlusion, will be diagnosed on the basis of symptomatology and other clinical and lab



ABDOMINAL ANGINA
PAIN AFTER EATING

DIARRHEA

MALABSORPTION SYNDROME

BOWEL NECROSIS

COMPLETE OCCLUSION
WITH INADEQUATE
COLLATERAL CIRCULATION

BYPASS GRAFT



THROMBOENDARTERECTOMY

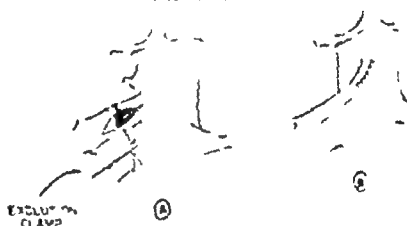


FIG 27 Gradual thrombosis of the superior mesenteric artery may give rise to abdominal or intestinal angina characterized by pain after eating. The relative ischemia may also result in a form of malabsorption syndrome. Abrupt complete occlusion will usually cause gangrene of most of the small bowel and the proximal part of the colon.

oratory findings of hepatic-gastric-splenic dysfunction plus aortogram.

Superior Mesenteric Artery

In contrast to the lack of specific clinical findings in the presence of celiac artery thrombosis occlusion of the superior mesenteric artery (Fig 27) may result in what are now recognized to be typical clinical findings. These symptoms which are due to ischemia and which may precede large scale infarction are sufficiently dramatic to afford an opportunity for diagnosis and effective surgical therapy prior to the onset of serious bowel necrosis.

General Considerations

The problem of mesenteric vascular occlusion was reviewed by DeMuth, Fitts and Patterson²⁶. The superior mesenteric artery supplies blood to the intestinal tract from the second portion of the duodenum to the midtransverse colon and this vessel is in effect an end artery. Anastomoses exist between the superior pancreaticoduodenal artery from the celiac axis and the inferior pancreaticoduodenal artery a branch of the superior mesenteric artery. The inferior mesenteric artery usually is not important in the blood supply of the small bowel but it may supply the small bowel in the presence of occlusion of the superior mesenteric artery—a finding which has been demonstrated by aortogram. The inferior mesenteric artery is commonly ligated (and is often already thrombosed) during resection of an aneurysm of the abdominal aorta. Even where there is acute occlusion of this vessel as in ligation for resection of

large bowel cancer, ischemic bowel necrosis is rare. Chiene's case, mentioned previously, represents an extreme example in which the blood supply to the bowel was supplied from somatic arteries in the presence of occlusion of the celiac axis, superior mesenteric, and inferior mesenteric arteries. Rob and Owen⁸⁰ reported ligation of thrombosed celiac axis and superior mesenteric arteries in resecting an aneurysm, with uneventful recovery of the patient. This again further emphasizes the extensive collateral circulation which can be developed. Thus the gradual occlusion of almost any mesenteric vessel may be tolerated.

Pattern of Arteriosclerotic Narrowing of the Celiac and Superior Mesenteric Arteries. In a necropsy study of 110 unselected aortas of patients ranging from 28 to 86 years of age, Derrick, Pollard, and Moore²¹ examined the incidence and pattern of arteriosclerosis of the arteries to the abdominal viscera. Gross anatomic observations were confirmed by roentgenologic studies after the aortas had been redistended with air. Forty-four per cent of the celiac arteries examined demonstrated some narrowing secondary to arteriosclerosis. Twenty-one per cent were found to have, at some point, a reduction of 50 per cent or more in the calculated cross-sectional area of the vessel. Narrowing of the superior mesenteric artery was found to be present in 37 per cent of the aortas examined. By and large, it was found that the area of the aorta just above the aortic bifurcation usually demonstrated the earliest changes of atherosclerosis and, in late cases, the most marked changes; the second area most frequently involved was the *ostia* of the abdominal visceral branches.

Clinical Findings

Types of Pain.^{47 48 49 50} In writing of abdominal pain of vascular origin in 1936 Dunphy⁵¹ pointed out that vascular disease was not then generally recognized as one of the causes of abdominal pain. The so-called abdominal or intestinal angina^{47 48} had been described for many years but lack of postmortem evidence had prevented its acceptance as a clinical fact. It was noted that Sir William Osler and others had favored the view that transient attacks of abdominal pain in arteriosclerotic individuals were manifestations of angina pectoris rather than of visceral arterial disease. It was felt in many quarters that there was no evidence of vascular pain in the abdomen apart from that due to actual gangrene and peritonitis secondary to terminal mesenteric vascular occlusion. Nevertheless Dunphy reported a case of a 47-year-old laborer who had entered the Peter Bent Brigham Hospital with recurrent abdominal pain of two months' duration which was definitely related to meals and occurred about one and one-half hours after eating. For a time the avoidance of heavy meals and the taking of a daily enema and mild cathartic had alleviated the pain but the severity of the attacks had gradually increased. On several occasions there had been slight diarrhea but no nausea or vomiting. Roentgen ray studies of the entire gastro-intestinal tract had been negative and the patient had been diagnosed from another clinic as having psychoneurosis. At the time of admission to the Peter Bent Brigham Hospital he was complaining of excruciating and constant abdominal pain associated with nausea and vomiting but without jaundice, diarrhea or melena.

On physical examination he was found to be well developed but had obviously lost considerable weight recently. There was tenderness to the palpation in the epigastrium, but there was no muscle spasm or abdominal distention. The leukocyte count was 20,000.

It was considered likely that the patient was exaggerating his symptoms. Although mesenteric thrombosis was believed a possibility, the long duration of symptoms appeared to make it unlikely and a tentative diagnosis of subacute cholecystitis was made. The patient died suddenly three days after admission to the hospital but, up until a few hours before death, no significant changes in the physical findings had been noted. Autopsy revealed occlusion of the celiac axis and the superior and inferior mesenteric arteries. Almost the entire small bowel and a part of the large bowel were gangrenous. It was felt inescapable that a gradual occlusion of the mesenteric arterial system by progressive thrombosis had been the cause of recurrent abdominal pain of two months' duration. Although the pain was severe, it was not sharp or localized, did not radiate, and was not associated with muscular spasm or exquisite tenderness of the abdominal wall. In the beginning the pain was definitely related to the ingestion of food and only later became constant. Dunphy concluded that "vascular pain" in the abdomen is a result of anoxemia of the intestinal wall and is a true visceral pain manifested through sensory neurones in the sympathetic nerves independently of the musculocutaneous pathways.

A much more comprehensive survey of the problem of embolism and thrombosis of the superior mesen-

eric artery was published by Klein²⁴ in 1921. In recognizing important publications even earlier than his, he quoted at length from the article of Reich²⁵ and summarized conclusions of earlier investigators very nearly as we know the picture of gradual superior mesenteric artery thrombosis today. In general they found that the pain was frequently severe, often colic like in nature. It was present in almost 85 per cent of the cases. At first the pain occurred only occasionally but later the intervals between episodes of discomfort were shorter and the pain gradually merged into that of peritonitis when gangrene of the bowel supervened. It was found that obstipation was usually present at some time during the course of the condition and that vomiting might occur occasionally being bloody. The occurrence of bloody stools was a most ominous sign when associated with the other symptoms though such stools occurred in only about 30 per cent of the cases. Diarrhea however was not uncommon and often preceded the obstipation. Abdominal tenderness was present in a large majority of cases and was frequently generalized. Whether or not the patient had fever depended upon the pathology present; the temperature was usually normal early but fever developed with the onset of peritonitis where bowel gangrene and perforation occurred. Although acute occlusion of the superior mesenteric artery might not always produce gangrene of the bowel, since collateral supply might preserve viability, far more commonly the occlusion of the superior mesenteric artery that was not fatal had developed slowly over a period of weeks or months, affording considerable time for collateral flow to develop.

Reich⁸⁴ had suggested the following grouping for different types of cases of mesenteric arterial occlusion (a) a *diarrheal group*, in which the diarrheal movements began at the outset and persisted throughout the course of the illness, or ceased with the onset of peritonitis. The movements occurred often and were profuse. At times the stools were bloody. (b) An *obstructive group* in which obstipation persisted from the onset of the pain to death, though occasionally diarrhea preceded it. Bloody vomiting occurring with this group was especially significant of serious disease. (c) A *mixed group*, where the two above groups were intermingled. Most commonly the diarrhea was followed later by obstipation. Less frequently the order was reversed. (d) A group with meager symptoms in which precise diagnosis was practically impossible.

It is more and more apparent that chronic arterial occlusive disease is usually due to atherosclerosis and rarely to thrombangitis obliterans.²

The Malabsorption Syndrome. Thus the fact that visceral ischemia may result in various types of abdominal pain has been realized for a great many years. However, only recently has general attention been drawn to the fact that relative ischemia of the bowel may result in a malabsorption syndrome.^{11, 19} In brief, by suitable studies it is possible to demonstrate impaired fat and d-xylose absorption,⁵ and examination of the stools may show undigested meat fibers.²¹ Shaw and Maynard²¹ reported two cases of atherosclerotic occlusive disease of the superior mesenteric artery in which surgical relief was accomplished by thromboendarterectomy. In one patient

acute thrombosis was associated with transient post operative malabsorption. In the other chronic thrombosis produced a picture of intestinal malabsorption, intermittent abdominal pain and weight loss, eventually leading to acute intestinal infarction. The diagnosis in this second patient was established preoperatively by aortography as it was in the case reported by Derrick Pollard and Moore.²¹

It is clear that in certain cases of abdominal pain or of defective intestinal motility or where unexplained malabsorption exists mesenteric arterial occlusion should be considered and in selected cases an aortogram should be performed.²²

Diagnosis

There can be no doubt that many cases of intestinal angina are still missed through failure to consider the possibility that mesenteric arterial occlusion exists. This manifestation of atherosclerotic occlusive disease will become more generally known in the next few years and in the future many cases will be discovered. This has been true of almost all types of occlusive disease that have been described. Therefore any patient with unexplained abdominal pain must be considered possibly to have inadequate arterial blood flow to the intestine. The presence of a *bruit* in the epigastrium is highly suggestive of arterial narrowing. When exploratory laparotomy for abdominal pain does not disclose a satisfactory explanation for the pain or even if it does the arterial pulsations in the celiac axis and the superior mesenteric artery should be examined particularly if the pulsations in the smaller mesenteric arteries to the bowel

itself are not vigorous to the naked eye. Again, any type of intermittent intestinal obstruction or obscure form of malabsorption should lead one to suspect the presence of mesenteric artery thrombosis.

When the possibility of acute or chronic mesenteric arterial occlusion exists, one has the choice either of exploring the patient or of performing an aortogram. The latter may be performed directly through the back or by passing a catheter up the femoral artery.¹ The femoral approach enables one to be more selective of the vessels visualized, since the catheter can be positioned under fluoroscopic guidance and little contrast medium is required. Yet, the direct translumbar approach is more rapid and is generally satisfactory.

The patient with sudden occlusion of the superior mesenteric artery, due to acute thrombosis or an embolus from the heart, will usually have dramatic symptoms and will require immediate operation. The distribution of bowel color changes will indicate occlusion of the superior mesenteric artery. Miller and DiMarc¹² reported a patient in whom a successful superior-mesenteric-artery embolectomy, with small bowel resection, had been performed with recovery of the patient. Mikkelsen and Zaro¹¹ reported a patient with intestinal angina whose condition was correctly diagnosed preoperatively and whose symptoms were relieved by removal of the thrombosis in the superior mesenteric artery and the aorta. In discussing their case, these authors pointed out that the diagnosis of intestinal angina will usually be primarily one of exclusion, though of course one might perform an aortogram and at least demonstrate that mesenteric

arterial occlusion was present whether or not this might be causing the symptoms of which the patient complained. It was considered unusual however to encounter an entity other than intestinal angina in which the distinct "food pain" relationship was not accompanied by the demonstration of other physical laboratory or x ray findings. Weight loss would undoubtedly stimulate a suspicion of occult neoplasia. If such a possibility existed careful exploratory laparotomy would exclude the presence of neoplasm and disclose the presence of mesenteric arterial occlusion. Actually they considered that carefully performed exploratory laparotomy was superior to aortography in some instances in arriving at the correct diagnosis of mesenteric arterial occlusion. In the patient they reported there was extensive thrombosis in the aorta itself and there was an absence of pulsations in even the smaller mesenteric arterial branches. There was a thrill in the hepatic and splenic arteries but an expansile pulsation was absent in both these vessels.

Management

The approach to the management of acute occlusion of the superior mesenteric artery will depend upon the stage of the process at which the patient is seen¹¹. If recent acute occlusion has occurred with severe abdominal pain and evidence of impending bowel necrosis such as one might readily suspect in a patient with atrial fibrillation who could have thrown an embolus to the superior mesenteric artery prompt exploratory laparotomy would be indicated. If the bowel were not already gangrenous—and many cases will be seen in the future in which there will yet

be time to save the bowel by restoration of pulsatile arterial flow—one will seek to expose the superior mesenteric artery at its origin from the aorta and to perform an embolectomy. Although in the past acute mesenteric occlusion has all too frequently been diagnosed only at autopsy, current preoccupation with the possibility of intra-abdominal vascular ischemia will unquestionably result in more prompt surgical intervention in patients with abdominal pain which could conceivably be due to such arterial insufficiency.

In contrast to the acute situation, the patient may of course present with abdominal angina or with bowel dysfunction, with or without malabsorption. In these cases the use of an aortogram or a selective visceral arteriogram should be considered. If mesenteric arterial occlusion is demonstrated, laparotomy with thromboendarterectomy or an arterial bypass graft may be employed to relieve chronic thrombosis and obstruction at the origin of the superior mesenteric artery from the aorta.

Approach to the Superior Mesenteric Artery Various surgeons have preferred different approaches to the superior mesenteric artery. Access to this vessel may be gained through the gastrohepatic ligament, through the gastrocolic ligament, or beneath the transverse mesocolon adjacent to the ligament of Trietz. We prefer the last. If it is desired also to expose the celiac axis, a thoracoabdominal incision may be more useful than the usual laparotomy incision. At times it may be advisable to reflect the peritoneum from the lateral abdominal wall and to approach the branches of the aorta from the retroperitoneal position, such as one might expose a

thoracoabdominal aneurysm for resection and grafting

Inferior Mesenteric Artery

Occlusion of the inferior mesenteric artery rarely results in gangrene of the parts of the left colon which it supplies. Nevertheless if the collateral blood supply is reduced by atherosclerotic narrowing of the superior mesenteric and hypogastric arteries necrosis of the descending colon may occur.^{21, 22, 23, 24} Clinical findings may include loose bloody stools, left sided abdominal pain and tenderness and perhaps a palpable mass usually associated with a shock like state. Prior to this however the patient may have experienced abdominal cramps and diarrhea due to relative ischemia. Chronic ischemia can produce scarring and thickening of the bowel wall with a loss of haustral markings. An excellent review of the blood supply which the colon receives from the superior mesenteric artery has recently been published.²⁵

OCCLUSION OF THE RENAL ARTERIES

One of the most recent developments in the field of arterial disease is the demonstration that essential hypertension is not infrequently due to atherosclerotic occlusion of one or both renal arteries.^{26, 27, 28} (Fig. 28) Such lesions are often remediable by thromboendarterectomy^{27, 28} or bypass graft techniques.²⁹ It is therefore necessary carefully to evaluate any patient who exhibits unexplained hypertension.²⁸

Pathophysiology In 1934 Goldblatt and his associates³⁰ demonstrated experimentally that a persistent elevation of systolic blood pressure could be produced

by means of renal ischemia. After this demonstration in animals a good many patients were found throughout the years whose hypertension was relieved

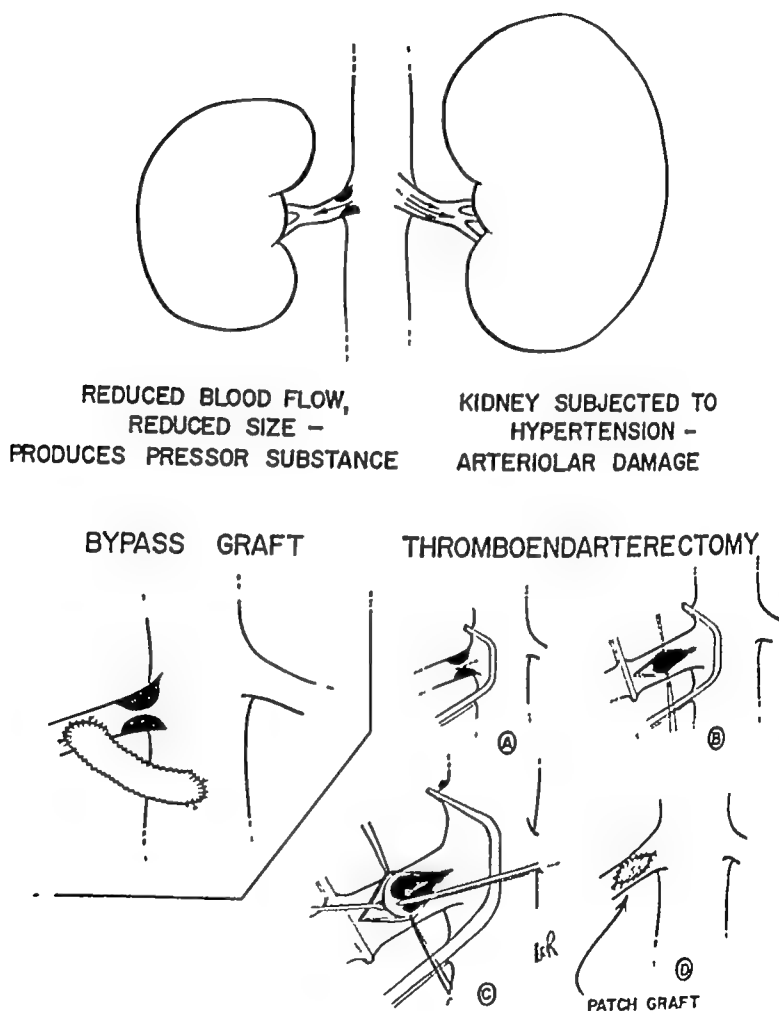


FIG 28 Renal ischemia due to atherosclerotic thrombosis may cause a form of "essential" hypertension. The occlusion is best relieved by either thromboendarterectomy or bypass graft. A patch graft can be useful in avoiding constriction of the lumen following arteriotomy.

by the removal of a "nonfunctioning" kidney¹⁰ It was assumed that the offending kidney was producing the pressor material¹⁰⁻¹² that caused the hypertension. In the years that followed there were sporadic reports of partial occlusion of renal arteries by atherosclerosis and thrombosis¹³⁻¹⁵ However not until recent years following frequent use of aortography has the relatively high incidence of the association of systemic hypertension and partial or "apparent complete occlusion of a renal artery been appreciated. In 1957 Poutasse and Dustan¹⁶ reported their studies involving 104 selected hypertensive patients examined in a two-year period (1955-56). Of these 30 were found to have unilateral or bilateral obstructive lesions of the renal artery. In most patients the lesions were considered to represent the primary cause of the hypertensive disease but some were felt to be aggravating a pre-existing hypertension. Most of the obstructing lesions were on the basis of atherosclerotic plaques and there were six instances of post-stenotic aneurysmal dilatation. Of the 30 patients who were demonstrated to have renal arterial occlusive disease 19 had either nephrectomy or corrective renal artery surgery which usually resulted in remission of the hypertensive vascular disease.

Morris and his co-workers¹⁷ have performed renal revascularization procedures on 52 hypertensive patients with renal artery stenosis. Eighty-one per cent of these patients developed normal blood pressure levels following operation. Nineteen per cent were unimproved but demonstrated some residual hypertension. In unilateral renal artery stenosis the kidney distal to the occlusive process often became the better

kidney following revascularization, from both functional and pathologic standpoints. The reason for this appeared to be that the kidney whose artery was stenosed was protected from the nephrosclerotic effects of the hypertension it produced, whereas the normally vascularized kidney was not so protected. In many of their cases the excretory urograms appeared normal prior to operation. In other instances, a kidney which did not exhibit excretory function on urogram preoperatively was normal in this respect following revascularization.

It has been emphasized by Poutasse and Dustan and by Morris and his associates that at the present time aortography or renal arteriography is the only dependable method of demonstrating occlusion of the renal arteries, despite various selective excretion tests which have been suggested. In discussing the basis for selection of aortography, Poutasse and Dustan emphasized that this procedure was not used in the routine investigation of hypertension, but was performed according to the following indications. First, any hypertensive patient, regardless of his age or duration of the hypertension, who showed unexplained disparity of the size or function of the kidneys by intravenous urography was so studied. It was found that disparities in kidney length of only one to two centimeters, or a slight delay in appearance of radiopaque medium on one side, might be indicative of renal artery occlusive disease. As a general rule, a kidney which was shown by intravenous urography to be nonfunctioning, but which was anatomically normal on retrograde pyelogram, was considered to have an obstruction of its vascular supply. Even so, important as such disparities in size and function

might be it was emphasized that the intravenous urogram could be entirely normal in patients with obstructive arterial lesions. Second it was felt that aortography was indicated in young patients who did not seem to have essential hypertensive disease since there was no familial history of hypertension or any other demonstrable cause.²² In the study reported eight hypertensive patients who were shown to have renal artery disease were less than 35 years of age and three were less than 20 years of age. It is of course now fully appreciated that coronary artery disease or lower aortic occlusive disease may occur in patients in the third or fourth decade of life. Third patients who were elderly and hypertensive but who suddenly developed accelerated or malignant hypertension were offered aortography.

Since the syndrome of malignant hypertension is a rare complication of essential hypertension in patients more than 55 years of age Poutasse and Dustan²³ emphasized that the development of such a complication should prompt a suspicion that the hypertension may be caused by renal arterial occlusive disease. Fourth it was believed that any patient whose long standing essential hypertension becomes abruptly more severe should have aortography to exclude renal artery occlusive disease. The possibility of occlusive disease was rendered even more likely if previous urograms had been done and later there was a definite change from the previous studies. Certain of these patients might develop hypertension after an attack of flank pain suggesting possible thrombosis of a renal artery with subtotal renal infarction.

It would appear therefore that perhaps the majority of patients with unexplained or essential hyper

tension should have arteriographic visualization of the renal arteries

Management of Renal Artery Occlusive Disease

A number of the problems involved in the management of renal artery occlusive disease have been mentioned above. Specifically, the presence of such arterial occlusion must be established, usually with an arteriogram. Once the occlusion has been demonstrated, one may relieve the obstruction near the origin of the renal artery from the aorta by either thromboendarterectomy⁸² or arterial bypass graft⁷³. Occasionally the involved kidney or a portion of it must be resected. Nevertheless, wherever possible every effort should be made to restore normal arterial flow rather than to excise renal tissue.

Most important, the relief of renal artery obstruction has commonly been followed by a return of the blood pressure to normal. This finding will serve as a tremendous stimulus to provoke a fresh analysis of, and a more aggressive attack upon, hypertensive cardiovascular disease.

OCCLUSION OF THE LOWER AORTA AND ARTERIES TO THE LOWER EXTREMITIES

A wide clinical experience now has established the fact that obstructing lesions in chronic atherosclerotic occlusive disease of the arteries to the lower extremities (Fig. 29) are frequently well localized and segmental in nature, having a relatively normal and patent lumen both above and below the level of occlusion^{10, 75, 110}. A general appreciation of the seg

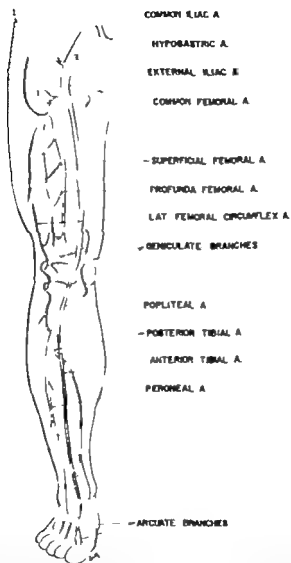


FIG 29 The surgical anatomy of the arteries to the lower extremity assumes increasing importance now that reconstructive arterial surgery is available

be associated with an opportunity to restore good blood flow to the lower extremities than is femoral occlusion largely on the basis of the fact that aortoiliac occlusion is usually associated with an extensive collateral circulation and in addition that the vessels being operated upon are larger and thus postoperative thrombosis is much less likely to occur. In other words, the surgeon would prefer to find that the ischemic disease of the lower extremities is due to occlusion high up at the bifurcation of the aorta rather than in the femoral artery below.

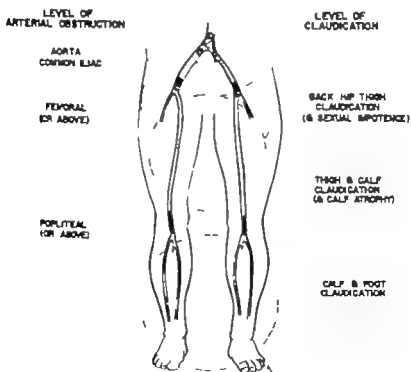


Fig. 30 The history and physical findings are usually diagnostic of the approximate level of arterial occlusion

Collateral Circulation. It was seen above that the collateral circulation and the rate of its development are highly important in determining the symptomatology which follows mesenteric arterial occlusion. Similarly, the collateral circulation in the lower extremities is highly important in determining the clinical findings which the patient with occlusive disease of the terminal aorta and more distal arteries will present. For example, acute occlusion of the lower aorta and both iliac arteries by a saddle embolus from the heart, perhaps secondary to recent myocardial infarction with mural thrombosis or to atrial fibrillation with clot in the left atrial appendage, may present with cold, almost white legs in which very little blood flow can be demonstrated, bilateral amputation may well be required if the embolus is not removed in time. In marked contrast, if the occlusion of the terminal aorta develops slowly over a period of weeks or months or years, the collateral circulation may be sufficiently extensive actually to produce weak pulses in the feet in the occasional patient. This collateral circulation not only has an important bearing upon the date at which the patient seeks medical advice regarding the pain of the intermittent claudication, it is also an important consideration when one selects the type of direct arterial attack to be employed in relieving the ischemia of the lower extremities due to occlusive disease. For every effort should be made to avoid dividing major collateral arteries in operating for occlusive disease of the lower extremities, and this will frequently mean the employment of an arterial bypass graft instead of resection of the occluded segments with use of a direct graft to replace the excised

arterial segment. Edwards²³ has pointed out that the effectiveness of collateral systems depends upon (1) the size of the anastomoses (2) occlusion patterns as regards collateral involvement (3) functional needs of the tissues supplied and (4) the general factors affecting blood flow and blood content. In discussing collateral circulation he points out that in at least one paired organ, the kidney, total necrosis of a single member is less harmful to the patient (less likely to produce hypertension) than survival of the organ with a reduced arterial circulation. Winblad and others¹⁶⁷ studied the etiologic mechanisms of collateral circulation using interarterial pressure tracings, arteriography, cinefluorography, tissue pO_2 and in the dog perfusion of a major artery distal to the occlusion with saturated and unsaturated blood. It was believed that the collateral circulation in the dog as measured by arteriography and distal pressure tracings, corresponded directly with the pressure gradient at the site of occlusion. Perfusion of the extremity distal to occlusion with saturated and unsaturated blood produced no appreciable effect on the development of the collateral circulation.

In his review of collateral circulation, natural and artificial, Learmonth²⁴ noted that the task of collateral circulation is to carry blood to the capillaries in quantity and at a high pressure. This is best accomplished when the alternative routes return blood to the main arteries distal to a block. The general measures considered in encouraging the development of collateral circulation include rest, anticoagulant drugs and the avoidance of local heat to the ischemic part. The collateral circulation could be concluded to be en-

couraged artificially by reflex vasodilatation, drugs, and occasionally by sympathectomy

General Diagnostic Considerations

Symptoms Whereas ten years ago necrosis of the tip of a toe associated with the pain of intermittent claudication^{9, 13} usually resulted first in a sympathectomy and later in amputation of the toe or, more likely, the leg at a level just above the knee, few physicians would now recommend amputation for minimal necrosis of a toe without first having established the probable level of arterial occlusion and whether or not reconstructive arterial surgery were possible to repair the arterial defect. Arteriography is usually essential in reaching such a decision. For this reason it is particularly important to emphasize here the symptoms of occlusive disease of the lower aorta and arteries of the legs. Digital ischemia so produced must be distinguished from that due to other causes (Fig 31)

The classic description of aorto-iliac thrombosis was recorded by Leriche in 1940 and came to be known as the Leriche syndrome⁶¹⁻⁶³. As he described it, the typical symptom complex occurred in men in the third to the sixth decades of life. Fatigue and weakness of the legs, with inability to maintain a penile erection, were emphasized as common complaints. Physical findings included an absence of arterial pulses in the lower limbs, muscle atrophy and pallor, but a relative absence of trophic changes in the toes. Subsequent to his description it became clear that claudication or weakness involving the hip area, in

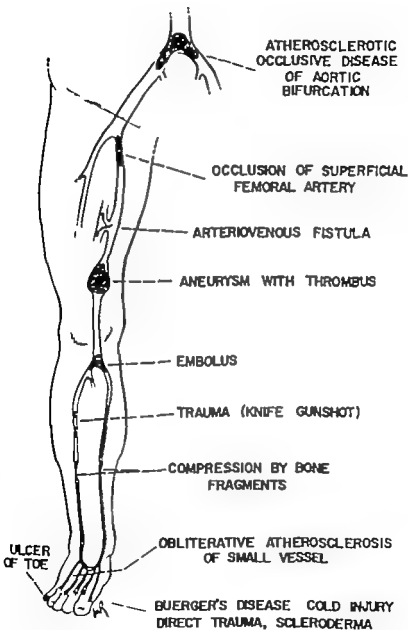


FIG 31 Many disease conditions may cause ischemia of a toe

cluding the low back and thighs, was a fairly common finding^{22, 33}

Despite the impetus which the dramatic concept of "Leriche's syndrome" imparted to arterial surgery, the clarity or sharpness of this syndrome has been somewhat dissipated by a vast amount of clinical experience with occlusion of the terminal aorta and the iliac arteries. The loss of distinctness of the clinical picture initially described has been due to variations in the frequency with which the group of clinical findings originally described have been found together²³. There are, of course, innumerable variations in the precise locations of sites of segmental occlusion,^{44, 50} and it is the specific tissue deprived of adequate blood supply which determines the clinical findings.

Neurologic diagnoses are not infrequently made when the underlying defect is arterial in nature. Gillilan and his associates³⁸ have drawn attention to certain types of occlusive disease which are particularly likely to be confused with nerve lesions. The findings of muscle atrophy associated with muscle weakness have been mentioned. In the presence of lower aortic occlusion severe pain on walking may develop in the buttocks, thighs, and legs, usually in this order. Fatigue of the involved muscles usually precedes the onset of frank pain. In total iliac obstruction the patient has pain beginning in the buttocks and extending into the thigh and into the calf on the affected side. This pain may bear a close similarity to pain secondary to nerve root irritation in the low lumbar and sacral levels, and may offer a difficult diagnostic problem in some instances. Partial iliac

obstruction may be associated with a less extensive group of findings. Obstruction of the deep femoral artery may result in pain in the anterior and medial aspect of the thigh in some cases. Obstruction of the superficial femoral artery or the femoro-popliteal artery results in the well known claudication pain occurring primarily in the calf.

Thus it is not uncommon for the neurologist to see patients whose underlying disease is due to arterial occlusion nor is it rare for the vascular surgeon to be referred patients for possible reconstructive arterial surgery who actually have pulses in their feet and whose underlying defect is neurologic rather than vascular. The vascular surgeon can usually exclude serious arterial disease if he finds relatively good pulses present in the feet and of course at all levels above this point including pulsations in the abdominal aorta.

The duration over which the symptoms have been present neither includes nor excludes the possibility of aortic or other arterial occlusive disease. We treated one patient with occlusion of the terminal aorta due to atherosclerotic thrombus who had known that he had weak or absent pulses in his legs for at least 14 years. He had progressively been able to find less and less exacting jobs from an exercise standpoint, to keep abreast of the diminishing arterial circulation in his legs. In reviewing 65 cases of chronic aorto-iliac thrombosis Beckwith and his associates⁴ found that in 30 per cent of the cases the patient had had intermittent claudication for one year or less. Seventy-seven per cent had been symptomatic for five years or less, and 91 per cent for less than ten years.

However, four of their patients had been symptomatic for ten years or longer.

Physical Findings Accompanying the slowly progressing distress consisting of either fatigue or actual pain in the calf, thigh, hip, or back produced by exercise are certain physical findings. The more distal pulses are of course usually absent. In the presence of complete terminal aortic thrombotic occlusion one will usually feel no femoral, popliteal, or foot pulses—though in the occasional case the collateral circulation may be sufficient to produce weak pulses at various levels. Relative ischemia due to partial aorto-iliac occlusion can be associated with a relatively normal aortogram.

The abdominal aorta is easily palpated through the abdominal wall in most patients who are not obese. Likewise, the iliac arteries can be palpated at most points from their origin at the aorta to the inguinal ligament, and the femoral pulses can be palpated below the inguinal ligament. The popliteal pulses are more difficult to detect than are the other pulses in the leg, though the popliteal pulses become much easier to find after one patiently practices and comes to know their location. Therefore, careful evaluation of all pulses from the aorta down to and including those in the foot is an essential part of any evaluation of the arterial blood flow to the lower extremities. Next in importance is an evaluation of the nutritional status of the skin and its appendages, as well as the evidence of the presence or absence of muscle atrophy. Marked trophic changes and atrophy in the feet are less common with aorto-iliac occlusion than with femoral or femoropopliteal occlusion.

Such trophic changes consist of decreased hair growth decreased or abnormal toenail growth thinning of the skin and subcutaneous tissue and reduction in circumference of the calf due to muscle atrophy. Any or all of these findings along with pallor or coldness of the foot and lower leg should prompt careful evaluation of arterial status.

A bruit occasionally associated with a thrill is a not uncommon finding over any partially occluded artery and the iliac and femoral arteries are not exceptions. We have frequently found a bruit and thrill over the partially occluded iliac or femoral artery in patients with occlusive arterial disease. In addition to the occlusive disease of the lower aorta and arteries to the lower extremities associated cardiovascular disease is quite common since atherosclerosis is of course a generalized process. Included among these other manifestations of vascular disease are hypertension renal insufficiency cerebrovascular disease coronary artery disease and at times visceral ischemia within the abdomen.

Aortography and Visualization of More Distal Arteries. Aortograms have been used extensively in the demonstration of occlusion of the aortic bifurcation (Fig. 32) and more distal arteries. The clinical circumstances will dictate whether aortic or femoral arteriography or neither is indicated in the given case. Since arteriography is not without an occasional serious complication there has been a trend toward the more conservative carefully selected use of this procedure. At the present time many do not employ aortography when physical findings indicate occlusion of the terminal aorta or the iliac arteries. Un



FIG 32 This aortogram revealed occlusion of the lower portion of the abdominal aorta

less there are major contraindications, one is going to perform a laparotomy to relieve the obstruction of the terminal aorta and of the iliac arteries, and it is not always necessary to subject the patient to the risk of translumbar aortography. One simply exposes

the lower aorta and the iliac arteries and determines from direct examination the extent of obstruction. Actually we no longer perform translumbar visualization even of aneurysms unless they are associated with occlusive disease or it is suspected that the aneurysm extends above the level of the renal arteries.

The most essential portion of arteriography in connection with management of occlusive disease of the vessels to the leg is found in the demonstration of an adequate run-off to the vessels supplying the lower leg. Thus the femoral artery is often exposed and if good back flow is not apparent a femoral arteriogram is performed prior to aorto-femoral bypass. For little of benefit would be accomplished by relieving aorto-iliac obstruction if there were large scale obliteration of the more distal arteries. In fact a bypass extending from the lower aorta to the common femoral artery might well become quickly thrombosed if there were no adequate run-off in the femoral artery distal to the point of anastomosis of the lower end of the graft to this artery.

Nevertheless the judicious use of aortograms and more distal arteriograms is a most important adjunct in the precise and effective management of ischemic disease of the lower extremities.

Other Diagnostic Measures. It is fair to say that most vascular surgeons doing a continuous volume of peripheral arterial surgery determine whether or not operative intervention is indicated in most patients by means of the clinical findings, selective arteriography and the use of the blood pressure cuff and the oscillometer. However certain additional measurements have been found useful upon occasion. These

include the measurement of skin temperatures,¹⁰⁵ the use of pulse registration²⁹ and related techniques¹¹

Aorto-iliac Occlusion

The surgical relief of occlusion of the terminal aorta and the iliac arteries is now commonplace^{19, 30, 31}. Although such an operation was suggested by Leriche⁶ in 1923, direct surgical attack to restore blood flow was not feasible at that time. Fortunately, for some years a combination of nonsurgical measures, with or without lumbar sympathectomy, did provide a considerable measure of relief in many patients with this condition.

It was pointed out above that the patient with aorto-iliac occlusion usually complains of slowly progressing distress in the calf, thigh, hip or back, usually induced by exercise and relieved by rest. In reviewing 448 cases of aorto-iliac occlusion treated by surgical attack, DeBakey and his associates¹⁹ found that the age range of those patients with complete aorto-iliac occlusion was essentially the same as those in which there was incomplete aorto-iliac occlusion. The youngest patient was 23 and the oldest 83 years of age, with the highest incidence in the fifth, sixth and seventh decades. Only 9 per cent of these cases were in females, emphasizing the well-known predominance in males. Of these 448 cases, 68 (18 per cent) had in addition occlusive disease involving peripheral arterial beds. This peripheral arterial occlusion was found more frequently in patients with incomplete aortic occlusion than in those with complete aortic occlusion. DeBakey and his group employed thromboendarterectomy, excision and grafting, or arterial

bypass graft or a combination of these. The indications for one or the other of these procedures were found to be dependent upon the extent and the nature of the occlusive process as well as upon certain systemic factors. Lumbar sympathectomy was considered a desirable supplemental procedure in cases in which there was associated peripheral disease with secondary narrowing or occlusion in the more distal arterial bed. Each of the three general methods of treatment was successful in restoring pulsatile circulation through the major arterial channels in a large percentage of cases. It was felt that this high degree of success confirmed the value of using a particular type of surgical attack in the given case to achieve optimal results with the method employed in the particular patient. Cardiac and renal disease constituted the major cause of death in the operative mortality of 27 per cent.

At the hospital of the University of Mississippi we have had a more modest but equally gratifying experience with the management of occlusive disease involving the terminal aorta and the ilio-femoral arteries. Once the symptoms have brought the patient to seek medical advice the diagnostic measures noted above are employed to determine the approximate level at which occlusion has occurred. If the occlusive process clearly lies within the abdomen aortogram is not usually employed. Laparotomy is performed and the level of occlusion determined by exposure of the aorta (Fig. 39). The occlusive process may of course extend up to the renal arteries in some patients whereas in others it is located just at the terminal aorta and its bifurcation. In still other instances the

process extends for some distance downward to include not only the common iliac arteries but at times also the external and the internal iliac arteries

If the occlusive process extends up to the level of the renal arteries, a clamp is placed briefly above the level of the renal arteries and the plug of thrombotic material is flushed downward or removed from the neighborhood of the renal arteries by incising the aorta transversely at a level about two inches below the renal arteries and performing thromboendarterectomy upon the segment between the level of this incision and the renal arteries. The occluding clamp is then moved to a level below the renal arteries and the remainder of the aorta is thromboendarterectomized, if this is desirable. If it is not, then a bypass

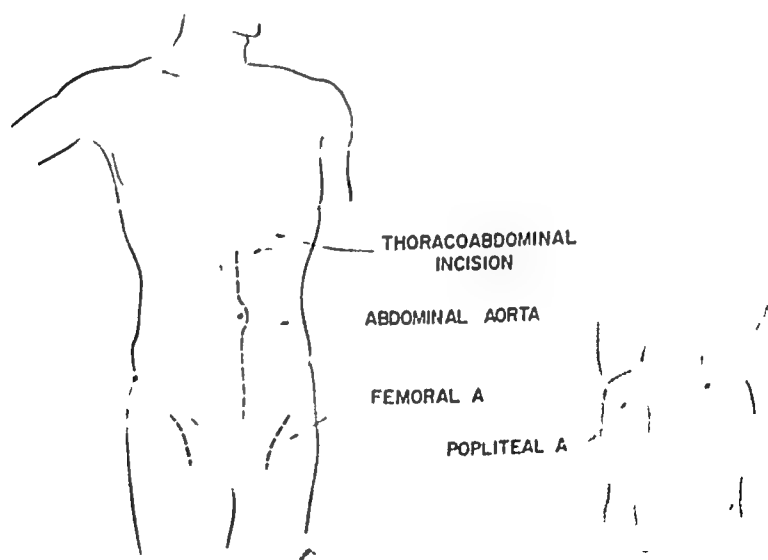


FIG. 33 The long mid line abdominal incision is suitable for exposure of the superior mesenteric artery and the more distal branches of the aorta. The aorta at the level of the celiac axis or above is often best exposed using a thoracoabdominal incision.

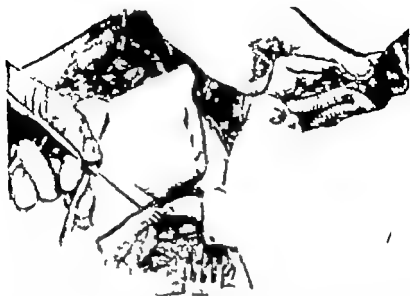


FIG. 34 An aorto-femoral bypass graft is shown. This patient had severe bilateral hip-thigh-calf claudication prior to the bilateral revascularization of the common femoral artery and its tributaries. Excellent pulses were restored in both feet.

graft is employed extending from the aorta to the common femoral arteries on either side (Fig. 34-35) and at times it is continued to the popliteal artery (Fig. 36). There is a distinct hazard in thromboendarterectomy of the aorta just below the renal artery for various authors have recorded renal failure due to embolization of portions of the thrombus to the renal arteries.²⁰ For this reason we have been extremely careful to dislodge as little of the thrombus as possible when performing thromboendarterectomy just below the renal arteries and we have taken the precaution of palpating for pulsation in each renal artery following this procedure.

Thromboendarterectomy for highly localized occlusion at the aortic bifurcation is usually quite satisfac-

tory However, when the occlusive process involves the terminal aorta and a considerable portion of each common iliac artery, the probability of a successful thromboendarterectomy without early postoperative occlusion by new clot is reduced Therefore, in general we have employed thromboendarterectomy for occlusion of the aorta itself and for short occlusions of the iliac arteries We have not employed thromboendarterectomy in recent months for occlusive disease

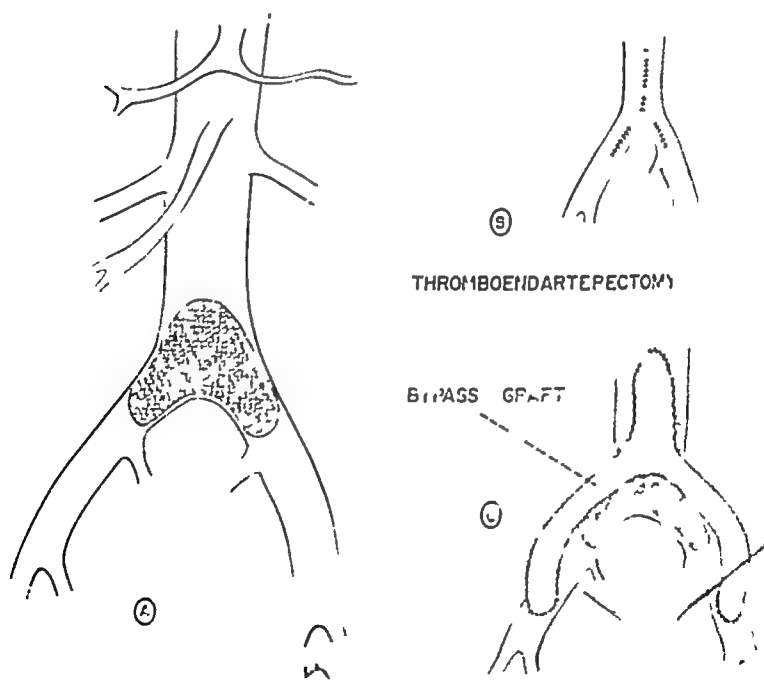


FIG. 55 Aortoiliac occlusion may be managed by thromboendarterectomy if the thrombosis is segmental and highly localized at the aortic bifurcation. If the thrombosis is extensive thromboendarterectomy is apt to result in early postoperative clotting and thus a bypass graft should be used.

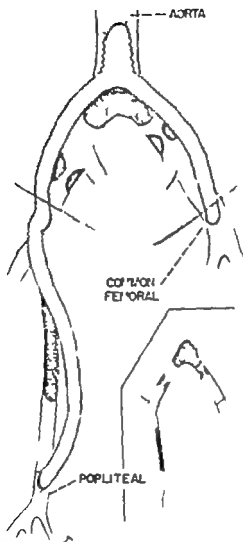


FIG. 36 The aorto-femoral bypass graft can be extended to include end-to-side anastomosis to one or both popliteal arteries.

of the femoral arteries or for occlusive disease of even the external iliac arteries. The aorto femoral bypass usually functions quite well and there is a lower incidence of early postoperative thrombosis.

Unless the occlusive disease of the lower aorta is associated with aneurysm formation, few surgeons still employ resection of the lower aorta and the aortic bifurcation with grafting^{10, 52, 106}. In the absence of aneurysmal dilatation, then, either thromboendarterectomy or arterial graft bypassing, or a combination of these two, comprises our usual form of management of occlusive disease of the lower aorta and the iliac arteries. At the present time we are using both Teflon and Dacron prostheses, though early reports indicate that the latter material may prove more satisfactory. The prosthesis usually extends from the anterior surface of the abdominal aorta above the level of occlusion to the common femoral or, in cases of femoral occlusion, to the upper popliteal artery. The diameter of the bypass graft prosthesis that is employed is usually almost twice the size of the femoral or popliteal artery to which it is being anastomosed in an end to side fashion. We prefer a continuous stitch of 4-0 arterial silk. A Y graft extending from the lower aorta to each common femoral artery is shown in Figure 31. This Y bypass graft immediately restored strong pulses in each foot, whereas preoperatively he had not been able to walk more than a few steps due to ischemia resulting from occlusion of the terminal aorta and the iliac arteries.

Occlusion of the Femoral and More Distal Arteries

All in all the patient with occlusion of the superficial femoral and more distal arteries is less likely to have an optimal result from surgical intervention than is the patient with occlusion at the terminal aorta and iliac arteries¹⁹. Nevertheless a great deal can be done for patients with occlusion of the superficial femoral artery²¹. As noted previously Roberts and Hoffman²² found that in about 30 per cent of patients who were admitted for possible amputation of the leg it was possible to perform suitable arterial grafting to relieve the ischemia. Most of their patients had occlusion of the superficial femoral artery. Thus arterial grafting may obviate the necessity for amputation in a significant percentage of cases with gangrenous or pre-gangrenous lesions of their extremities. Bypass grafting in the leg is accompanied by a higher incidence of early and late postoperative thrombosis than is thromboendarterectomy or bypassing of aorto-iliac occlusion. Nevertheless amputation should not ordinarily be undertaken for arterial occlusion unless arteriograms have demonstrated that the patient would not be helped by a grafting procedure. If the occlusion is distal to the popliteal artery grafting procedures are at the present time not suitable to relieve the ischemia in most subjects.

While the physical findings in the presence of aorto-iliac occlusion are usually sufficient to permit exploratory laparotomy without the performance of an aortogram it is essential to use arteriograms in determining the levels of occlusion and the collateral circulation in arterial occlusive disease in the leg itself.

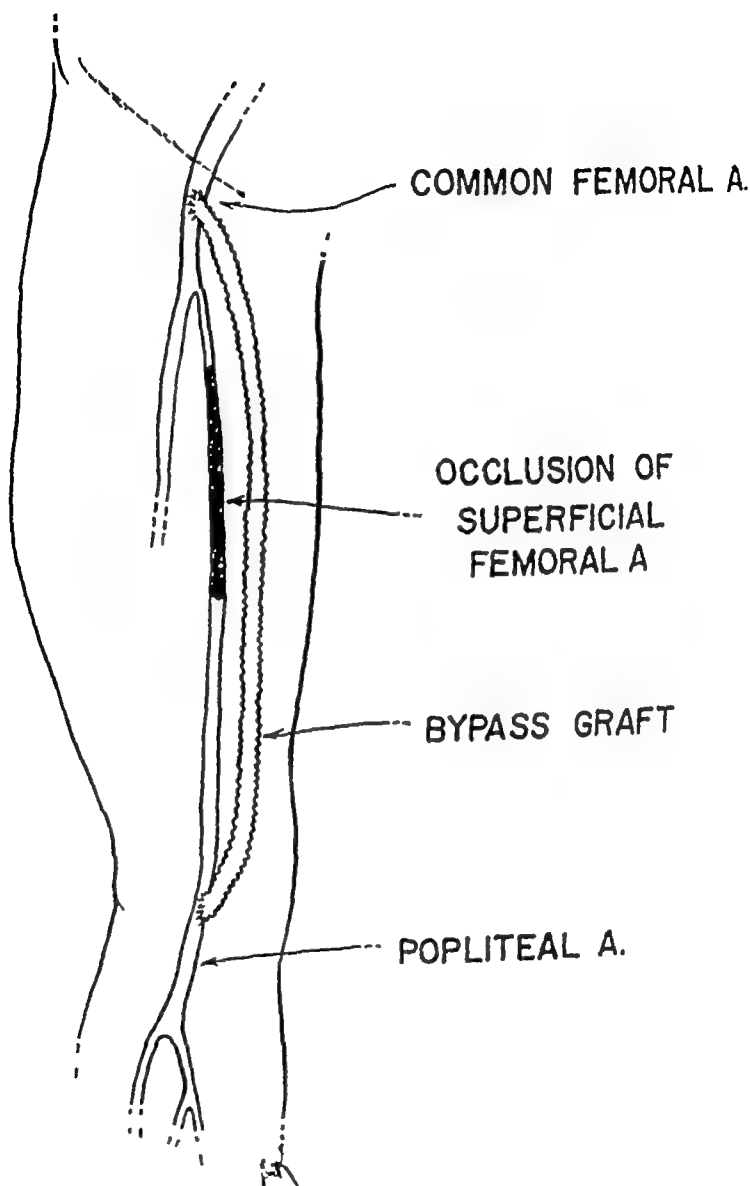


FIG. 37 The bypass graft which extends from the artery to a common femoral artery is less likely to thrombose than one which extends from a common femoral artery to the corresponding popliteal artery

Accordingly if the common femoral pulsation is present and good on the side involved indicating that obstructive disease does not exist in the vessels situated in the abdomen the common femoral artery is formally exposed by incision on the operating table under spinal or general anesthesia and an arteriogram is performed (The arteriogram can of course be performed percutaneously prior to operation) This will demonstrate whether or not there is a suitable re-entry for a run-off tract coming back in below the level of the occlusion If the radiopaque medium re-enters the main channel of the upper popliteal artery below the level of the femoral occlusion the patient is considered suitable for a bypass graft extending from the common femoral artery to the upper popliteal artery (Fig 37) At times as noted the length of the segment involved by the occlusive process may be no more than 1 cm in this circumstance one may occasionally do a thromboendarterectomy successfully or perhaps more commonly a short bypass graft (for sizes see Fig 38) We have employed both procedures with satisfaction Nevertheless it should be emphasized again that thromboendarterectomy^{12 44 51 54} for occlusive disease below the level of the common femoral artery is rather unsatisfactory and we seldom employ it at the present time

Actual resection of the involved segment of the femoral artery is now rarely performed for two reasons first, this type of grafting procedure is perhaps associated with a somewhat higher incidence of thrombosis than is the bypass graft and second—and more important—the excision of such a segment of the artery destroys collateral circulation in many instances

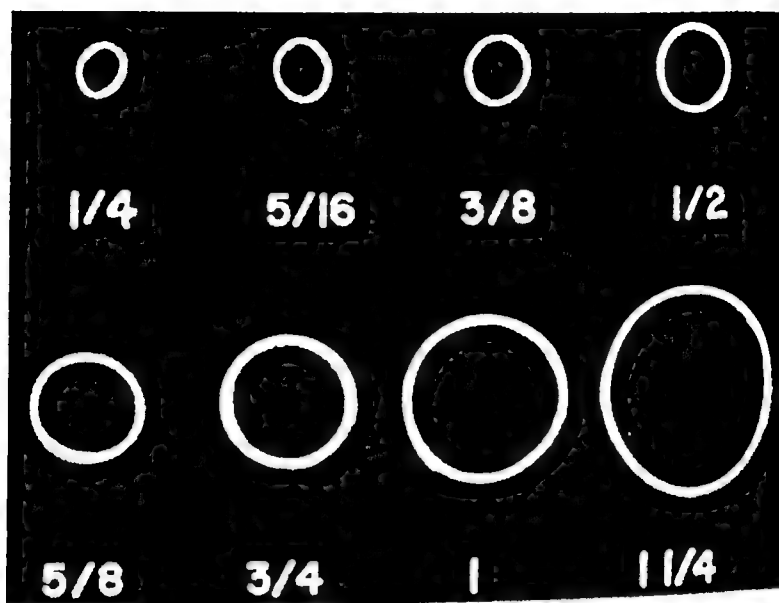


FIG 38 In selecting the proper size prosthesis at surgery, it is helpful to have rings of various sized prostheses mounted on a piece of cardboard for inspection. In this way one avoids unwrapping several sterile grafts before the one of the most satisfactory size is found. Although the diameter in inches is of course inscribed on the outside of the sterile wrapping, actual visualization of grafts of various sizes achieves more accuracy in selecting the proper size than does one's memory regarding how large a graft of say, one inch actually is.

If following operation a bypass graft should become thrombosed, the patient is not worse than he was previously, if the surgeon has not sacrificed collateral circulation. On the other hand, if the collateral circulation has been molested in the process of actual resection of the diseased segment with grafting, early thrombosis of the graft may result in loss of the leg when the patient had been able to walk into the hospital. A fundamental principle of direct arterial sur-

gery in the leg is not to leave the patient in a worse condition than he was when he came to the hospital. This objective is best achieved by employing the arterial bypass graft with or without lumbar sympathectomy.

In summary, whereas there is admittedly a significant incidence of early and late thrombosis in the bypass grafts which must extend from the aorta to the common femoral or to the popliteal artery (Fig 37) the results of the treatment of patients who require such grafts are usually gratifying. Most subjects are relieved of ischemic pain and their exercise tolerance is greatly increased. If occlusion of a femoropopliteal graft should occur as it will in a number of patients, there is no contraindication to performing a second bypass graft usually with equally satisfactory results as compared with those following the original graft. Moreover, since the operation involves only the vessels in the extremity and one does not need to open the abdomen, the presence of associated cerebral, cardiac or renal disease is not quite as serious a contraindication to operation as it would be if the aorta and iliac arteries were involved.

Adductor Canal Thrombosis Dunlop and Santos¹¹ have emphasized that most of the thromboses of the femoral artery occur in the adductor canal. In a review of 41 femoral arteriograms they found that 76 per cent showed an obstruction of the superficial femoral artery at this level. It was their conclusion that the predilection for occlusion to develop in the superficial femoral artery at this level is the result of local trauma caused by the adductor fascia and tendon as the artery is compressed between these structures and

the femur. Fixation of the artery was considered to prevent longitudinal expansion and to reduce the output through the collateral circulation. Unroofing of the adductor canal, mobilization of the artery and excision of the thrombosed segment was recommended in suitable cases.

While this concept is an interesting one, resection and grafting of the femoral artery itself probably is not now done by most vascular surgeons. We have preferred to use a bypass graft from the common femoral to the popliteal artery.

Anterior Tibial Muscle Compartment Claudication with Incomplete Arterial Occlusion. It has occasionally been noted that pulses in the feet may be present before exercise but may disappear during and early following exercise. In this connection Jones⁶⁶ has described anterior tibial muscle compartment claudication with incomplete arterial occlusion. In a patient he reported there was anterior tibial compartment muscle claudication in which both dorsalis pedis and posterior tibial pulses, present on initial examination, had disappeared after exercise. These signs and symptoms were completely relieved by end arterectomy of a short segment of the partially occluded femoral artery.

Arterial Bypass Below the Knee. In an effort to afford pulsatile flow to the foot of the patient whose proximal popliteal artery was not suitable for bypass grafting, Morris and his associates⁷¹ anastomosed the lower end of a femoral bypass graft to the popliteal artery just below the knee. Although this is not often done, since in many such patients the proximal popliteal artery is suitable for distal bypass anastomosis.

combined approach to the common femoral artery and the distal popliteal or posterior tibial artery is sometimes necessary in the treatment of occlusions of the popliteal artery or popliteal aneurysms. The above authors approached the popliteal artery through an anteromedial incision below the knee. This incision was made with the patient supine in combination with femoral and abdominal incisions where indicated. Nine instances were reported in which below the knee arterial bypass graft was used in eight patients. All patients in whom a bypass graft had been extended across the popliteal space were instructed not to flex the knee beyond 90 degrees. It has been shown that "squinting may cause complete kinking of a bypass graft which has been rendered more rigid" by being situated in the tissues

OCCLUSIVE DISEASE OF THE TERMINAL AORTA AND MAJOR ARTERIES IN THE LEGS

It is not possible here to review all the considerations which are of importance in the surgical management of patients with occlusive disease of the lower aorta and the iliac and femoral arteries. Nevertheless, certain general statements are warranted in this highly important and genuinely promising field of vascular surgery. First there is no question but that clinical experience and judgment are most advantageous in reducing the incidence of nonfatal and even fatal complications. Inasmuch as the patients are frequently elderly and have multiple associated diseases of the cardiovascular system as well as other organs at times fine judgment is required to deter

If Priscoline is used, it may be given four times daily in a dosage of perhaps 50 mg. As regards exercise, the patient should be cautioned to stop walking and rest when his legs become tired or actual intermittent claudication develops. If there is a tendency to swelling of the feet, he should lie down during a portion of each day to reduce the edema and to thus facilitate tissue oxygenation.

It should be borne in mind that further development of collateral circulation is possible in most instances, if the extremity can be made to survive an acute episode of threatened gangrene, this state may be followed by a considerable period of relative comfort due to improvement in the collateral circulation. Cranley¹⁵⁻¹⁷ has stressed the importance of clinical grading of the severity of obliterative arterial disease as a guide to treatment and prognosis.

Sympathectomy If frank and major gangrene has not developed by the time the patient is admitted for consideration of possible amputation because of distal arterial occlusion in which no reconstructive arterial surgery is possible, sympathectomy should be offered in most subjects. For a time we were quite pessimistic regarding the value of lumbar sympathectomy in the management of atherosclerotic occlusive disease involving the lower extremity, but a number of recent cases have restored a measure of confidence in the value of this procedure in certain selected patients.²⁰ Of course, most surgeons have performed sympathectomy for atherosclerotic occlusion for many years, particularly prior to the availability of more satisfactory techniques with which to restore pulsation to the lower

A number of good reviews regarding the value of sympathectomy in large series of cases have been published 27 32 67 7 8 9* 93 100 101

In brief in the patient who has only minimal necrosis of a portion of a toe or perhaps an ulceration of the lower leg or intermittent claudication in whom there is no possibility of performing a satisfactory arterial bypass sympathectomy should usually be performed. Actually it is our practice to take the patient to the operating room and under either spinal or general anesthesia to expose the femoral artery and perform an arteriogram for visualization of the femoral artery and its major branches including the popliteal artery. If it is demonstrated to be feasible to perform a femoro-popliteal bypass this is done usually without the additional performance of a sympathectomy. However if a bypass is not feasible a lumbar sympathectomy is performed through an abdominal *lateral abdominal* or lumbar approach. Most patients will obtain a measure of relief from intermittent claudication or even rest pain following sympathectomy. Needless to say such relief is not to be favorably compared with that which is achieved when a satisfactory bypass can be constructed but it nonetheless does prevent gangrene in many instances and it least increases the length of time before an amputation of some type may be required. Furthermore it should not be forgotten that a sympathectomy which spares the patient pain and postpones amputation for even a year or two years is often almost as effective as the bypass graft which may thrombose fairly early following operation.

Summary and Conclusions

1 The pathophysiology, diagnosis and management of atherosclerotic occlusive disease involving the abdominal aorta, its major visceral branches, and the arteries to the legs have been reviewed

2 Celiac artery thrombosis is not often diagnosed clinically unless it is associated with occlusion of the superior mesenteric artery. This is because the organs supplied by the celiac axis have either a dual or an extensive collateral blood supply or, in the case of the spleen, because the function of the tissue in question can be sacrificed without serious physiologic disturbances

3 Superior mesenteric artery occlusion can produce a typical food-pain type of abdominal angina at times prior to late ischemia so severe as to cause bowel gangrene and peritonitis. Relative small bowel ischemia can result in a form of malabsorption syndrome. The ischemia can often be corrected by means of either thromboendarterectomy or bypass graft

4 Inferior mesenteric artery thrombosis rarely eventuates in gangrene involving the left colon unless the superior mesenteric artery and the hypogastric arteries are also occluded

5 Renal artery thrombosis has long been known to result in systemic hypertension in the rare case. Recently, however, the widespread use of aortograms has disclosed that this ischemic mechanism exists in many more hypertensive patients than was previously realized. The finding of a small kidney on one side with reduced ipsilateral function on differential studies, should prompt an aortogram for visualization of the renal arteries in selected patients whose

hypertension is not otherwise satisfactorily explained. Following surgical correction of the renal ischemia the blood pressure commonly declines to normal.

6. A wide and quite successful clinical experience with the relief of aortic, iliac and femoral arterial occlusive disease has been amassed. Almost no leg should now be amputated for intermittent claudication, rest pain or minor necrosis of the foot until clinical and arteriographic studies have excluded the possibility of successful thromboendarterectomy or by-pass grafting. Surgical considerations involved in aorto-iliac and femoro-popliteal arterial reconstruction have been considered in some detail. Lumbar sympathectomy combined with meticulously executed conservative measures can often provide much relief where restoration of pulsatile flow through arterial surgery is not possible.

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CHAPTER 5

Aneurysms

FEW surgical achievements of modern times have been more generally acclaimed than has the successful management of arterial aneurysms. These lesions have been a source of fascination and challenge to physicians throughout the ages¹ but not until the past decade has genuinely effective excisional treatment been available. Patients with this hitherto hopeless condition can now often be offered a new lease on life.

Many surgeons contributed to these recent accomplishments but perhaps the most important developments were first the demonstration by Cross and his co-workers²⁹⁻³⁰ in 1918 that coarcted segments of the thoracic aorta could be successfully replaced with arterial homografts and second the demonstration by Dubost and his associates that an aneurysm of the abdominal aorta could be resected and replaced with an aortic homograft²²⁻²⁶. Following these advances many surgeons throughout the world contributed to the rapid further development of this excisional treatment for aneurysms, the most outstanding single group being that of DeBakey and his associates¹⁴⁻²³. At the present time aneurysms of all portions of the aorta and its major branches have been successfully resected and replaced with either homografts or with fabric materials such as Nylon, Teflon or Dacron.

The various factors involved in the surgical treatment of aneurysms of the aorta and its branches will be discussed under the following headings

A General Considerations

1. Etiology and Definitions
- 2 Signs and Symptoms of Aneurysms
- 3 Diagnostic Measures
- 4 Prognosis Without Surgery
- 5 General Principles of Surgical Attack upon Aneurysms

B Aneurysms at Different Levels of the Aorta and Its Branches

- 1 The Innominate, Carotid and Subclavian Arteries
- 2 The Thoracic Aorta
- 3 The Thoraco-abdominal Aorta and Its Visceral Branches
- 4 The Abdominal Aorta Below the Renal Arteries
- 5 The Iliac, Femoral and Popliteal Arteries

GENERAL CONSIDERATIONS

Etiology and Definitions

Aneurysms may be either saccular or fusiform or both, and they can be caused by a variety of agents which weaken the vessel wall. In the past it was generally believed that *syphilis* was the most common cause of aneurysm formation, and certainly this disease still results in many instances of arterial wall damage. Thoracic aneurysms are more often due to lues than are abdominal aneurysms. But even so, the incidence of syphilitic aneurysms has steadily de-

clined until now most aneurysms of the abdominal aorta and perhaps the majority of those in the thoracic aorta are due to *arteriosclerosis* in one form or another. Of particular interest are the relatively uncommon patients with cystic medial necrosis in some of whom the classic collateral findings of Marfan's syndrome are present.³ The so-called *congenital aneurysms*⁴² are due also to a congenital defect in the structural composition of the wall of the artery, usually of the media. There may be a dearth of elastic fibers or of muscle fibers. A *traumatic aneurysm* may represent a *true aneurysm* in which at least one layer of the wall of the vessel is still intact, or a *false aneurysm* (pulsating hematoma) in which the wall of the vessel has been disrupted and the aneurysm consists of encapsulated hematoma. The false aneurysm may or may not pulsate depending upon whether the blood contained in the false sac has become clotted or not. *Poststenotic dilatation* or aneurysm formation has been explained by Holman⁴³ on a physical basis as shown in Figure 40. In brief the



FIG. 40 Mechanism of poststenotic dilatation (after Holman⁴³). The eddy formation with increased lateral pressure which occurs distal to the constricted portion of the artery is believed gradually to weaken the vessel wall and produce poststenotic dilatation. It is possible that some atherosclerotic aneurysms of the lower abdominal aorta are produced by a similar process.

lateral force produced by eddy formation just distal to a point of arterial constriction is believed gradually to weaken the wall of the vessel and dilatation eventually results. It is possible that certain atherosclerotic aneurysms may be produced in a similar manner. A *mycotic aneurysm*^{43, 81} is one which originates on the basis of infection either within or surrounding the vessel. The condition is particularly likely to occur in patients who have had left-sided endocarditis⁶⁵. In a collected series of 382 mycotic aneurysms in 217 patients, Stengel and Wolfenb⁷⁰ described aneurysms of intravascular septic origin as being due to (a) those developing from infected emboli which lodge in the vessel lumen or in the vasa vasorum, (b) those due to the deposition of microorganisms directly on the intima of the vessel or in the vasa vasorum and (c) those caused by extension of infection from the aortic or pulmonic valves, in which case the proximal portions of the aorta and pulmonary artery respectively are involved.

A *dissecting aneurysm* is one in which the continuity of the intima is disrupted and the force of the blood dissects an intramural channel. These lesions most often develop in the proximal portion of the aorta. A torn atherosclerotic plaque may be found at the origin of the defect, and at times the intramural channel may rupture back into the main channel of the aorta below. The term *arteriovenous aneurysm* is really a misnomer, unless there is an arterial aneurysm present in addition to the simple arteriovenous fistula—which frequently is the case.

Signs and Symptoms of Aneurysms

Aneurysms may give rise to symptoms and signs which mimic a great many different disease processes. The clinical findings produced by an aneurysm will of course depend upon its location and size relative to the region of the body in which it arises. In general the symptoms produced by an aneurysm are due to the compression of surrounding structures. An aneurysm of the inferior thyroid artery may encroach upon the recurrent laryngeal nerve to produce hoarseness.²⁴ Those of the arch of the aorta or the adjacent great vessels may compress the trachea or bronchus produce cough or dyspnea; hoarseness may result from paralysis of the recurrent laryngeal nerve; dysphagia from compression of the esophagus or pain from erosion of bony structures. Compression of the phrenic nerve can paralyze the hemidiaphragm on the involved side. And of course the larger aneurysms may suddenly rupture and permit hemorrhage which may or may not be immediately fatal.

Abdominal aneurysms are easy to detect in thin individuals but in obese subjects they frequently are not diagnosed until rupture has occurred. The pain which the patient had prior to rupture may have been attributed to intestinal obstruction (for which exploratory laparotomy had been negative) or to arthritis of the spine, or to a ureteral stone with pain radiating down into the scrotum in the presence of a leaking aneurysm. We have operated for ruptured abdominal aortic aneurysm when more than one previous laparotomy for attacks of abdominal pain had revealed no pathology. Our pathologist subsequently reported that study of the resected aneurysm revealed that

several different times in the past it had leaked into the retroperitoneal space and then sealed off. As a further example of how confusing the symptoms and signs caused by a ruptured abdominal aneurysm can be, a patient was admitted to our urology service because of pain radiating from the left flank down into the scrotum. Only when he went into shock some days following hospitalization and a mass abruptly became palpable in the lower abdomen was the correct diagnosis made. Prompt surgical intervention resulted in successful resection of a large aneurysm with homografting in this obese man. Other complications of abdominal aneurysms have included rupture into the alimentary tract with massive G.I. bleeding,⁸⁵ rupture into the inferior vena cava with the creation of an aorto-caval fistula,^{27, 38} and severe erosion of the spine.

In view of the increasing incidence of aneurysms in our aging population,⁹⁰ the possibility of such a lesion should be considered in any patient with unexplained abdominal or thoracic pain (Table 2).

The history and physical examination will usually disclose aneurysms of the neck, extremities, and lower abdomen. Those situated in the thorax, however, must usually be diagnosed with x-ray. The possibility of aneurysm should be considered in the differential diagnosis of most mediastinal masses. Even a paraspinal "tumor" may represent an aneurysm,⁸⁴ and the vascular nature of the mass is usually best confirmed with appropriate angiography. However, we have considerably reduced the frequency with which we use arteriography for the demonstration of aneurysms that are otherwise obviously present. The pul-

Diagnostic Measures

TABLE 1. Some Signs and Symptoms of Aortic Aneurysms

A Thoracic Aneurysms

- 1 Hoarseness due to recurrent laryngeal nerve compression
- 2 Dyspnea and cough due to tracheobronchial compression
- 3 Dysphagia due to esophageal compression
- 4 Pain due to compression and erosion of bone
- 5 Venous distention (e.g. superior vena caval syndrome) due to compression of the great veins.

B Abdominal Aneurysms

- 1 Pain
 - a May occur in back due to erosion of the vertebrae
 - b May simulate other lesions such as pancreatitis intestinal obstruction or penetrating peptic ulcer
 - c May radiate from left flank into scrotum if aneurysm ruptures with left retroperitoneal hematoma
 - d Intermittent claudication of legs if aneurysm becomes filled with clot.
 - e May rupture into alimentary tract.
- 2 Pulsating Mass
- 3 Laboratory and clinical evidence of massive hemorrhage in absence of hematemesis melena hemoptysis or hemothorax

sating mass is exposed and after control of the artery proximally and distally has been achieved the aneurysm itself is attacked in whatever manner seems most appropriate. Actually the confirmation of an aneurysm is not usually difficult once the possibility has been suspected. Those of the aortic arch are best demonstrated with an angiocardioqram. This is also true for the descending aorta in many instances

Aneurysms of the abdominal aorta are readily demonstrated by means of an aortogram, if this is necessary. Perhaps the most common use of an aortogram in this instance is to identify the level of the aneurysm, that is, to determine whether or not it extends above the visceral arteries—renal, superior mesenteric and celiac. At other times one needs to exclude par

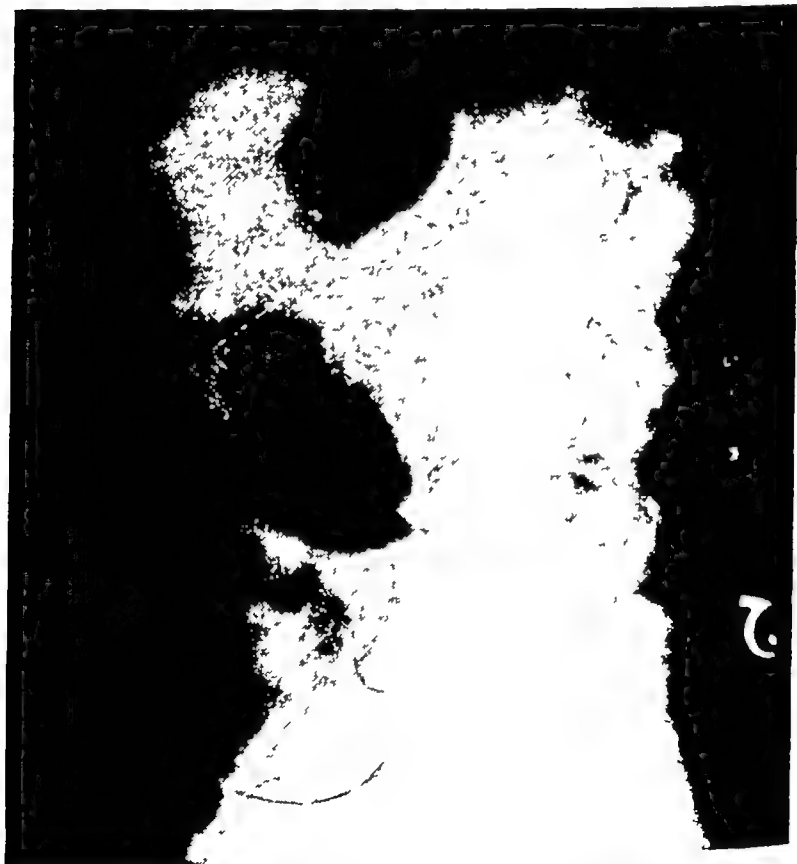


FIG. 41 Aneurysm of the abdominal aorta. Aneurysms can often be visualized roentgenologically because of calcium deposits in the wall of the vessel. In this lateral view of the abdomen the calcified wall of the aneurysm was clearly outlined. The P-A view frequently is equally helpful, but at times the calcific deposits overlie the spine and are thereby obscured.

tial occlusion of weakly pulsating femoral arteries distal to the aneurysm. Frequently the aneurysm itself can be readily visualized by means of plain P A and lateral films of the abdomen due to calcium deposition in the wall of the aorta (Fig. 41).

Prognosis Without Surgery

The ultimate fate of the patient with a large aneurysm of a major artery is about as poor as that of the patient with a highly malignant tumor which cannot be completely excised. Both lesions will usually prove fatal. In a study of 633 cases of saccular aneurysms of the thoracic aorta Kampmeier²² found that the average duration of life after the onset of symptoms was less than one year. In a similar study of 78 cases of aneurysm of the abdominal aorta he found that the majority had died within six months of the onset of symptoms usually due to rupture of the aneurysm.²¹ The prognosis of patients with aneurysms of the abdominal aorta however is somewhat better than that associated with aneurysms of the thoracic aorta. Estes²⁰ studied 102 cases and found that one third of the patients with aneurysms of the abdominal aorta died within a year after the diagnosis was established mostly from rupture of the aneurysm. He concluded that only 10 per cent of these patients at the age of 65 years had a normal life expectancy. Ghedman, Ayers and Vestal²³ studied the necropsy reports of 96 aneurysms of the aorta, iliac and other abdominal branches. The survival time appeared to coincide with other necropsy studies. Thirty per cent of the patients were dead within one month of symptoms, 75 per cent were dead within six months and 80

per cent were dead in less than one year. The survival rate in this group is much lower than in the group of patients in which an aneurysm is found incidentally. These workers noted that patients with a syphilitic abdominal aneurysm frequently had a second aortic aneurysm (64 per cent of 14 patients). Forty-nine per cent of the patients with abdominal aneurysms died from a vascular rupture. Only 4 per cent of patients with aneurysms died from a disease which was apparently entirely unrelated to their aneurysms or its underlying cause. Seventy-two per cent of all aneurysms 7 cm. or more in diameter were ruptured. On the other hand, less than 18 per cent of the aneurysms less than 7 cm. in diameter were ruptured. Hypertension was present in 47 per cent of the patients with arteriosclerotic aneurysms, and 35 per cent showed definite evidence of previous myocardial infarction. Colt¹² also found that rupture of the aneurysm was the most common cause of death in individuals with these lesions.

The prognosis associated with arterial aneurysms involving peripheral arteries is of course better than those involving the aorta, due to the greater possibility of successful surgical intervention should the aneurysm rupture. Nevertheless, one can look upon most aneurysms as potentially fatal lesions, for if successful surgery cannot be performed the patient may well lose his life. Exceptions to this are represented by aneurysms completely filled with thrombus, leading to distal ischemia but not to exsanguinating hemorrhage.

Thus the indications for surgery are comprehensive and relatively few contraindications will be found

other than critically impaired general health due to brain heart, kidney liver or other disease. And even in these circumstances additional experience with the resection of aneurysms of the lower abdominal aorta has rendered the morbidity and mortality rate sufficiently low that most patients in even fair physical condition can survive the procedure.

General Principles of Surgical Attack Upon Aneurysms

The successful approach to resection and grafting of aneurysms is accomplished by careful adherence to a relatively few well-established surgical principles. First preparations for adequate blood replacement must be made. There must be available enough compatible blood to meet any contingency which might be occasioned by rapid and massive hemorrhage. When the aneurysm is located in an extremity one can by the use of tourniquets and pressure virtually always prevent exsanguination. When the aneurysm involves the thoracic or upper abdominal aorta however there may be sudden hemorrhage prior to successful control of the aorta above and below the level of the aneurysm and one must always have available at least two venous routes for the rapid infusion of blood. Second there must be adequate exposure of the field. The skin incision should extend at least from one end of the aneurysm to the other. Third tapes should be passed around the artery both above and below the mass prior to any attack upon the aneurysm itself. One can never know when bleeding from the aneurysm may be precipitated, and thereafter it can prove difficult rapidly to identify and clamp the

artery entering and the artery leaving the aneurysm without risk of injuring adjacent veins, nerves and other structures. It is easy to identify and to secure the artery at a distance above and a distance below the aneurysm, for here inflammatory reaction occasioned by the aneurysm is minimal or absent and planes of cleavage have not been obscured. Once the main vessels have been secured, the patient will not exsanguinate from bleeding into the aneurysm through small collateral vessels such as the intercostal or lumbar arteries or collateral vessels in the extremities. Fourth, the assistants, instruments, and arterial replacement prostheses available should be appropriate to the nature of the aneurysm which is to be resected. It is now rarely permissible to ligate major vessels in the resection of an aneurysm, for a prosthesis should be inserted. Although ligation may not precipitate gangrene in an extremity, it certainly will not leave the patient with a normal blood flow to that extremity, and intermittent claudication is likely to be present postoperatively.

ANEURYSMS AT DIFFERENT LEVELS OF THE AORTA AND ITS BRANCHES

Now that the basic tactics of successful aneurysm resection have been considered, the salient features of the management of aneurysms occurring at different levels in the body will be reviewed.

The Innominate, Carotid and Subclavian Arteries

Aneurysms of the innominate,⁴⁶ carotid^{54, 77} and subclavian arteries are relatively common, and our group has operated upon aneurysms at these vari-

ious levels. With the exception of traumatic aneurysms which will be discussed in Chapter 7 these lesions are most frequently due to either atherosclerosis or syphilis. They occur more often in the elderly for it is in this group that the ravages of atherosclerotic vascular disease are the most extensive. The lesion usually presents as a pulsating mass which may or may not be painful and tender and which causes symptoms chiefly by pressing upon surrounding nerves, veins, the esophagus or the trachea. Hoarseness is a not uncommon accompaniment of aneurysms situated adjacent to the thoracic inlet for at this point the recurrent laryngeal nerve may be compressed on the left or as it passes upward from around the aorta and on the right it may be stretched where it passes around the subclavian artery. Likewise it is not rare for the patient to have a Horner's syndrome on the involved side since the sympathetic chain may be damaged by the expanding and pulsating mass. Occasionally the mass may not be pulsating and it may be mistakenly diagnosed as a solid tumor. This is due to the fact that the blood within the aneurysmal sac has clotted. Aneurysms of the innominate artery may not be apparent for some time because the innominate dilatation must be fairly large to be detected above the thoracic inlet. Moreover a prominent innominate or subclavian artery may represent tortuosity of the vessel rather than aneurysm formation. The origin of the innominate artery lies almost directly posterior to the manubrium of the sternum. Frequently the patient has had pain or other symptoms which prompted a chest x ray revealing a mass high in the mediastinum situated anteriorly. All

such masses should be considered to represent possible aneurysms until proved otherwise, but in our experience the most common mass in the superior mediastinum anteriorly has been the substernal extension of a goiter. If no thyroid mass is palpable in the neck, the chances are increased that the anterior superior mediastinal mass is vascular in nature. Other relatively common anterior mediastinal masses are dermoid cysts, pericardial cysts and thymomas, but with the aid of chest roentgenograms and fluoroscopy the radiologist is usually able to be fairly specific regarding whether or not the mass is an aneurysm.

Most subclavian and carotid aneurysms are readily available to palpation, and the diagnosis is not difficult unless the blood within the aneurysmal sac is clotted. These aneurysms may rupture, compress respiratory passages, cause difficulty in swallowing and, on occasion, interfere with the blood supply to the brain on the involved side, distal embolization of parts of intra-aneurysmal clot may occur.

Management. Satisfactory exposure of aneurysms of the innominate artery and the proximal portions of the subclavian arteries is best achieved by a sternal splitting incision which is taken into the third intercostal space on the involved side. One then gains control of the vessel both proximal and distal to the aneurysm. When this has been done the extent of the aneurysm and its relationship to surrounding structures can be determined. If it is a saccular defect with a relatively short neck, it may be possible to excise the aneurysm and to oversew its base or neck. Otherwise it must be resected and a graft inserted.

The most serious physiologic consideration under

these circumstances is the state of the blood supply to the brain during the period of occlusion⁴⁴ if the aneurysm is to be resected and a graft inserted. For a time hypothermia was employed to diminish the risk of cerebral ischemia and hemiplegia during the period of occlusion of the carotid flow on one side or the other in the resection of aneurysms. More recently however there has been a general trend towards the use of an arterial bypass of some type. Such shunts have been employed to extend around the aneurysm and in other instances have been passed through the aneurysm to extend from below to above it to maintain flow through the common carotid artery while the aneurysm of the innominate artery or of the subclavian artery was being resected. Of course if the innominate artery is involved the shunt will have to extend from the aorta to the carotid artery distally. It is not difficult to anastomose a Teflon tube either temporarily or permanently to the ascending aorta and to the carotid artery distal to the site of the aneurysm. In this way flow is maintained to the involved side of the brain while the aneurysm is being resected and a prosthesis inserted. By exclusion of a portion of the wall of the aorta and a portion of the wall of the carotid artery distal to the aneurysm the prosthesis can be anastomosed end-to-side both above and below with blood flow meanwhile being preserved through the aorta and both carotid arteries. Actually in perhaps 75 per cent of patients it is safe to clamp the common carotid artery for perhaps 30 minutes and resect an aneurysm but the facility with which bypasses can now be constructed renders even a 25 per cent risk of hemiplegia too great a

price to pay for mere convenience. It has been conclusively demonstrated that aneurysms of the innominate, subclavian, and common carotid and internal carotid arteries⁹ can be successfully resected and blood flow restored with a low mortality rate if blood flow to the brain is maintained by judicious measures and by the avoidance of emboli from the aneurysm to the arteries of the brain.

In the management of aneurysms of the subclavian artery distal to its origin from the innominate on the right, or from the aorta on the left, one need not establish bypasses, for the temporary occlusion of blood flow through a vertebral artery on one or the other side is rarely likely to result in hemiplegia due to inadequate blood flow through the basilar artery to the brain. Furthermore, the collateral arterial supply to the upper extremity is so great that actual ligation of the subclavian artery is uncommonly associated with serious after effects—though this should not be done where it can be avoided. Thus far the use of prostheses in replacing segmental occlusive disease or aneurysms in the upper extremity has not been conspicuously successful, due to thrombosis of these grafts. Nevertheless, relatively little experience has been gained thus far in replacing the more distal portions of the subclavian artery and the axillary artery with grafts, and further experience will undoubtedly improve the results of grafting these vessels.

Little comment need be directed toward the management of aneurysms of the carotid artery by preliminary slow occlusion with various types of clamps or by double ligation of the carotid and excision of the defective segment. These measures were employed

prior to the availability of suitable materials and techniques for the construction of shunts and they are now rapidly disappearing from the surgical armamentarium used to treat extracranial aneurysms.

The Thoracic Aorta

The most difficult aneurysms to manage are those arising in the thoracic aorta and particularly those involving the aortic arch. The principal reasons for this are, first, that the lesion is situated close to the heart and second, that it involves arteries which supply the brain. In resecting the aneurysm one must protect the heart from excessive strain due to occlusion of the aortic outflow tract and he must protect the brain from ischemia. Saccular aneurysms with a short neck can at times be excluded, excised and the neck oversewn.^{2, 3} However, relatively few such aneurysms are encountered and most aortic aneurysms must be treated by resection and grafting. Numerous patients with aneurysms of the entire aortic arch have now been successfully operated upon.^{13, 16, 17} By means of almost any desired combination of tubes for bypassing temporarily occluded arteries, virtually any accessible artery of significant size can be resected and a graft inserted.

Etiology and Types of Thoracic Aneurysms *Further Comment*

Syphilitic and Atherosclerotic Aneurysms. Although it was indicated previously that an increasing incidence of aneurysms of the thoracic aorta due to atherosclerosis is being noted, most of the reports of successful resections of the aortic arch have involved

patients whose aneurysms were most likely due to syphilis, though doubtless with an atherosclerotic component. The serology should always be carefully evaluated in any patient who has an aneurysm, and antiluetic therapy should be administered preoperatively if the serology is positive. It has been our experience that syphilitic involvement of the aorta is likely to be extensive. Furthermore, it is not uncommon for aneurysms involving the thoracic aorta to be multiple. That is, there may be general aneurysmal dilatation of the aorta from the heart all the way to the diaphragm, or there may be separate aneurysmal bulges at different levels. Nevertheless, many aneurysms are localized in a relatively short segment of the aorta, and these can be excised using the pump oxygenator or suitable bypass shunts on a temporary or permanent basis.

Cystic Medial Necrosis Including Marfan's Syndrome. The importance of Erdheim's cystic medionecrosis^{28, 62} in the genesis of aneurysms of the thoracic aorta has been emphasized by Bahnson and Nelson.⁵ This pathologic condition has been demonstrated in patients with dissecting aneurysms, in those with spontaneous rupture of the aorta,⁴² to a slight degree in "normal" aortas,⁶⁰ and in cases of Marfan's syndrome.^{45, 83} In fact, it is one of the major features of Marfan's syndrome,⁷⁸ in which patients also exhibit arachnodactyly, dislocated lenses, and a generalized defect in the ground substance.

Cystic medial necrosis may be an especially prominent cause of aortic aneurysms in young and middle aged subjects. A particular feature of such aneurysms when they occur in the ascending aorta is aortic in-

sufficiency due to dilatation of the aorta just distal to the valve. In some patients with this condition the aortic cusps are essentially normal and when the aneurysm is resected and the normal diameter of the proximal aorta restored competency of the aortic valve may also be restored.³

Thoracic Aneurysms Due to Trauma Trauma due to penetrating or blunt injuries is an important cause of aneurysms of the thoracic aorta. These will be discussed in more detail later (p. 339) but it should be noted here that gunshot wounds, knife injuries and even blunt trauma in the rapidly decelerating automobile or airplane accident can result in pulsating hematomas of the thoracic aorta. Furthermore occasionally a suture line may bleed as following resection of a coarctation of the aorta and here too an aneurysm may form which can at times be successfully resected with a second anastomosis of the aorta.⁴

Treatment of Aneurysms of the Thoracic Aorta

The principles previously outlined for the management of aneurysms in general apply equally well to the management of aneurysms of the aortic arch and the descending thoracic aorta. Again one must avoid circulatory overload upon the heart due to prolonged occlusion of the ascending aorta or the aortic arch. If this is not done heart failure will rapidly ensue. In fact, during resection of coarctation of the aorta situated at or just below the level of the origin of the left subclavian artery the heart will occasionally become dilated if the occlusion time is prolonged; this is especially likely to occur if there was significant blood flow through the coarcted segment prior to

cross-clamping of the aorta. Nevertheless, most patients can withstand occlusion of the coarcted aorta even above the level of the left subclavian artery up to 30 minutes, and of course they can readily withstand occlusion of the aorta below the origin of the left subclavian artery if the stenosis of the aorta at the level of the coarctation has been virtually complete. The reason for this is that the patient has established collateral circulation throughout his life. However, it should be borne in mind that a considerable amount of collateral circulation is interrupted as the muscles of the chest wall are incised in performing thoracotomy. Moreover, it is often necessary to divide one or two large intercostal arteries to achieve sufficient exposure to permit resection of the coarcted segment with end-to-end anastomosis. And in addition to the sacrifice of the collateral circulation and the intercostals at the aorta, one may find that a considerable volume of blood has been flowing through the coarcted portion of the aorta, this possibility is rendered even more likely by the absence of rib notching in older patients and the absence of dilatation of the intercostal arteries. In such subjects the risk of excessive cardiac strain and spinal cord damage during aortic occlusion should be borne in mind and the period of aortic occlusion should be minimized. Some surgeons have employed hypothermia⁴¹ to reduce the risk of aortic occlusion, while others have employed a bypass graft. Nevertheless, neither of these protective technics is required for successful resection of coarctation of the aorta in most patients. In contrast, it is absolutely essential that the ascending and proximal arch of the aorta not be

occluded for more than 1 minute or so in the course of resection of an aneurysm of the arch unless either the pump oxygenator or tube bypass is used. Cardiac overload was a cause of failure in early attempts at resection of aneurysms of the arch in addition to the fact that cerebral ischemia often resulted. More recently either the pump oxygenator in the case of aneurysms of the ascending portion of the aortic arch⁸ and various types of bypass shunts¹⁰ in the case of aneurysms involving the arch with its vessels to the brain have been employed successfully both to avoid cardiac overload and to continue adequate oxygenation of the brain during the period of aortic oc-



FIG. 42. Aneurysm of the distal aortic arch. The symptoms produced by most unruptured aneurysms are due to compression of surrounding structures. *Left* The trachea is seen to be displaced to the right by the large syphilitic arteriosclerotic aneurysm of the distal portion of the arch of the aorta. The patient had experienced increasing dyspnea. *Right* The esophagus is shown to be displaced to the right and posteriorly.



FIG 43 Bypass prosthesis The aneurysm of the aortic arch shown in Figure 40 has been resected following the preliminary insertion of a temporary-permanent Teflon bypass extending from the ascending aorta to the descending thoracic aorta below the level of the aneurysm. Clamps were applied to the aorta immediately proximal and distal to the aneurysm, the defect was excised and the ends of the aorta were oversewn. The left subclavian artery was the only major artery arising from the arch which was involved by the aneurysm.

clusion required for resection and grafting. Aneurysms of the descending portion of the arch and below (Fig 42), can readily be excised, since one can place a bypass prosthesis from the ascending aorta to a point below the aneurysm in the descending thoracic aorta (Fig 43). One can then either insert a prosthesis in replacement of the resected portion of the aorta, or he can elect to close the proximal and distal stumps of the aorta, leaving the temporary-permanent bypass graft as the channel of flow from the ascending aorta to the descending thoracic aorta (Fig 41).

In summary, aneurysms of the ascending aorta must

be managed by means of the pump oxygenator to prevent cardiac overload and to oxygenate the brain while the aneurysm of the ascending aorta is being resected. The blood is removed from the right atrium or the vena cava and is returned to the aortic system

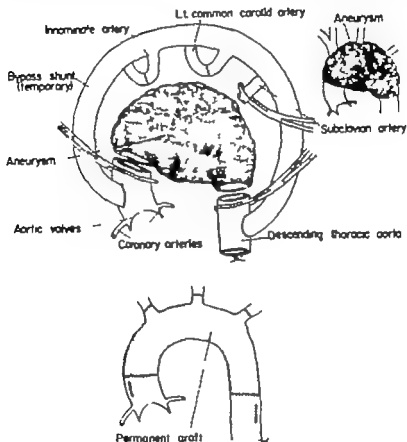


FIG. 44 The major steps used in the total resection of the aortic arch are depicted. First a temporary bypass is placed to avoid excessive back pressure upon the heart and to continue arterial flow to the brain and the distal portion of the aorta. Next the aneurysm is resected and a permanent graft is inserted. The temporary bypass is then removed. At times the temporary graft may be left as a permanent bypass, the ends of the aorta being oversewn following excision of the aneurysm.

by means of a catheter introduced through a femoral artery into the lower abdominal aorta. If the aneurysm of the ascending aorta also involves the vessels of the arch, one must use not only the pump oxygenator but also some type of bypass to permit blood flow to the brain during the period when the sites of origin of these vessels from the aneurysm must be occluded. This is done by anastomosing the shunt from the distal portion of the aortic arch to a point on the carotid arteries which is distal to the site at which one wishes to occlude them at their origin from the aneurysmal mass. An important point in the resection of thoracic aneurysms is that of avoiding injury to the phrenic nerves, for pulmonary ventilation in these older patients is often a major problem in the postoperative period. Too, since wide thoracic incisions have been made, often including median sternotomy and a trans-sternal component, the diaphragmatic portion of the respiratory effort is very important.

Saccular vs Fusiform Aneurysms Further comment should be accorded the possibility of management of saccular aneurysms of the thoracic aorta by the simple expedient of cross-clamping the base of the aneurysm adjacent to the aorta and then closing the aorta at this point with resection of the aneurysm. This is a genuinely feasible procedure in some patients. Unfortunately it does not occur as often as one might wish, but it does avoid the extensive procedures of using the pump oxygenator and a variety of bypass shunts which are required frequently for the resection of fusiform aneurysms of the proximal aorta. Bahnson² was one of the first to report an unusually successful group of patients treated in this way. It has

since been used by many surgeons on many occasions and it has been used by us. One should emphasize that the aorta and the arteries generally are usually affected by atherosclerosis in the presence of aneurysm and a palliative type of surgery is all that is possible in many cases. Therefore there is every justification for the exclusion and excision of an aneurysm by lateral aortorrhaphy when the opportunity for this presents as it infrequently does.

Dissecting Aneurysms

Until very recently the dissecting aneurysms of the aorta, usually arising in the ascending portion of the arch or just distal to this point, were considered to represent hopeless situations⁶¹⁻⁶³. Various sporadic reports of operative attempts to ameliorate the condition had been published but it was not until the report of DeBakey, Cooley and Creech²¹ that a genuinely effective and planned attack upon these distressing lesions was presented and executed (Fig. 45). It was pointed out that unlike the saccular and fusiform aneurysms for which excisional therapy is readily feasible, dissecting aneurysms of the aorta constitute a quite different problem.¹⁹ First the dissecting aneurysm is not often sufficiently well localized to permit the definitive application of excision and grafting. Second the dissecting aneurysm usually arises in the proximal aorta in the region of the aortic arch and most often in the ascending aorta.²⁷ The condition is fatal in the majority of cases frequently within 24 hours of the onset of pain. In his analysis of 143 collected cases Shennan⁷⁸ found that death occurred within 24 hours in 58 per cent and in one day

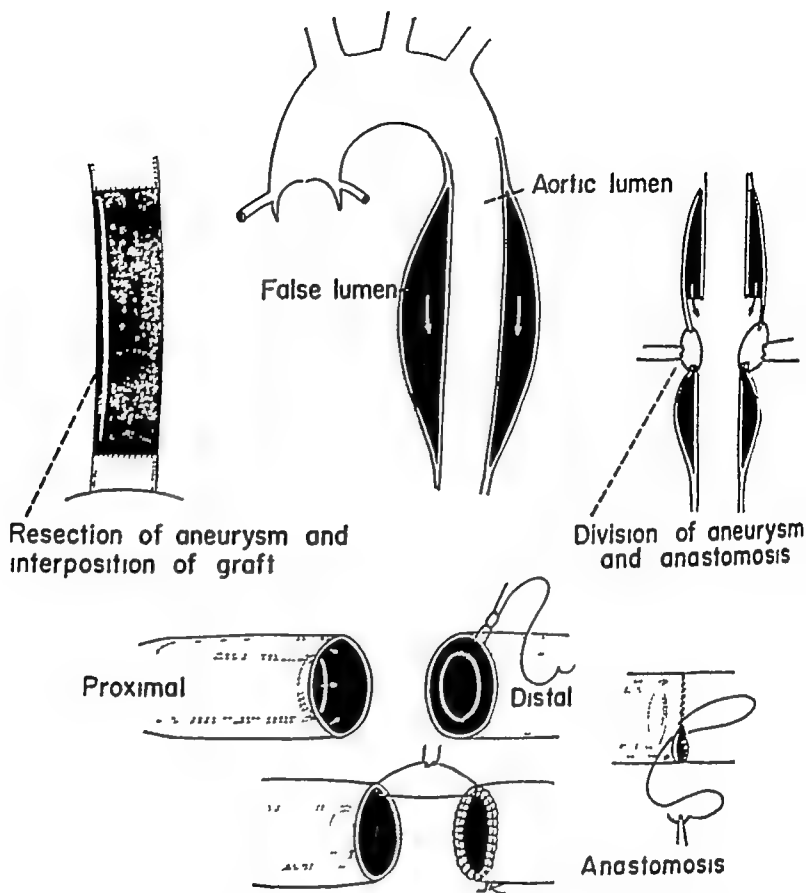


FIG 45 A dissecting aneurysm of the aorta is produced when disruption of the intima permits the force of the blood to dissect intramurally, usually beginning in the arch. At times a continuous double-lumen flow is produced when the blood stream ruptures back into the main aortic channel below. Two principal methods of surgical treatment have been devised. First, one can divide the aorta and close the intramural defect distally, returning the blood flowing through the wall of the proximal aorta to the main aortic lumen. Second, one may elect to resect a portion of the involved aorta and interpose a graft. Inasmuch as the prognosis is extremely poor without surgical intervention, operative attack is indicated in most cases where the diagnosis can be established with reasonable certainty.

to one week in 26 per cent. In the cases recorded by Weiss, Kinney and Maher²⁰ only 10 per cent survived for a significant length of time. Levinson, Edmeades and Griffith²¹ found that 36 per cent of their patients died within 48 hours of onset, 37 per cent within three to 60 days and only 25 per cent with the chronic or so-called healed form of the disease survived three months to eight years. DeBakey and his associates²² concluded that the condition is rapidly fatal in from 75 to 90 per cent of cases, affording every justification for bold surgical intervention as soon as it is diagnosed.

Diagnosis of Dissecting Aneurysm of the Aorta In our experience the diagnosis of dissecting aneurysm of the aorta is not always easy to make. Characteristically the patient is supposed to experience an excruciating pain beneath the sternum or in the back which progresses downward. However, frequently the pain does not progress downward and further more it is often difficult to distinguish between a dissecting aneurysm and an acute myocardial infarction. Therefore on history and physical examination alone one hesitates to perform a thoracotomy on a patient who may not have a dissecting aneurysm of the aorta. Fortunately certain roentgen studies are helpful.²³⁻²⁵ First a widening of the aortic shadow can frequently be demonstrated. This is particularly likely if a previous chest film is available to compare with one taken following the onset of pain. Second a double aortic shadow may be seen on angiocardiography²⁶ and the lumen of the main channel of the aorta may be irregularly constricted due to the intramural hematoma. Very occasionally one may be able

to demonstrate the flow of the radiopaque medium through both the main channel of the aorta and through the false intramural channel. However, more often the medium cannot be demonstrated to flow through the wall of the aorta. The presence of a "double shadow"—the appearance of the radiopaque medium flowing through the main aortic channel and the portion of the diameter of the aorta through which medium is or is not flowing and which represents the channel of dissection by the hematoma—is particularly helpful. Yet, in a number of cases a dissecting aneurysm has been proved at operation when it could be only suspected preoperatively, the surgeon opened the left hemithorax for the resection of a thoracic aneurysm and found also a dissecting component. The patient whose aorta is shown in Figure 44 had a dissecting aneurysm as well as two aneurysms of the aorta proximally and the dissection had begun at the base of the more distal aneurysm.

The reports in the literature do not indicate how many patients were explored who did not prove to have a dissecting aneurysm of the aorta. We performed thoracotomy in one patient in whom the attending cardiologist and radiologist had diagnosed a dissecting aneurysm, but the aorta was normal in every way. Fortunately the patient survived.

Management The successful surgical treatment of dissecting aneurysm of the aorta has now been reported from a number of clinics.^{10, 21, 74, 86, 87} Our group operated upon a patient with dissecting aneurysm of the aorta (Fig. 46) which presented as an expanding hematoma in the abdomen and was thought initially to represent a ruptured abdominal

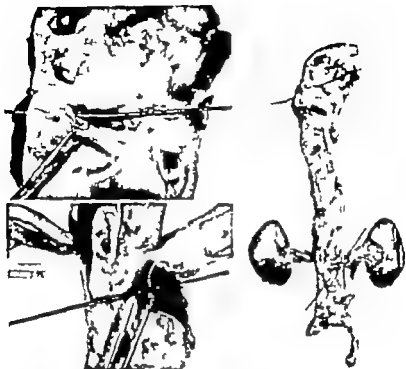


FIG. 46 Dissecting aneurysm of the thoracic aorta. At the upper left is shown the point of rupture of the intima at the distal extension of a shallow saccular aneurysm; the probe indicating this defect is shown again on the right. A second but smaller saccular aneurysm was situated more proximally. At the lower left the second probe has been passed through the aortic origin of the left renal artery, indicating that the vessel was compressed by the intraluminal hematoma; this probe is shown again in full length view of the aorta on the right. The patient was admitted to the hospital with a rapidly expanding abdominal mass. A dissecting aneurysm beginning in the thoracic aorta was suspected but laparotomy was performed to control the hemorrhage which was found to be arising from the ruptured splenic artery. Further hemorrhage did not occur but the patient died rather suddenly about two days later. The intramural dissection extended from the aortic arch to the external iliac arteries. (From Hardy J. D. *Pathophysiology in Surgery*. Baltimore: Williams and Wilkins, 1958.)

aneurysm. However, at laparotomy it was eventually discovered that the hemorrhage was arising from the celiac axis and, particularly, from the splenic artery. The dissecting aneurysm had dissected out along the splenic artery, rupturing through the adventitia, and this was the site of the hemorrhage. It was assumed that the patient did have a dissecting aneurysm which had arisen probably in the thoracic aorta, but the patient's condition was so poor that it was decided simply to ligate the splenic artery proximal and distal to the point of hemorrhage and to divide it between the ligatures. Since control of the hemorrhage had been accomplished, the abdomen was closed. The patient expired two days later from no truly obvious cause. He did not bleed again, and he had not been oliguric long enough to develop a serious uremic state. The blood pressure was markedly elevated until very shortly before he went into sudden collapse and died. The use of antihypertensive drugs was considered but was postponed, since he had previously been in shock due to blood loss into the abdomen. Autopsy revealed that the dissection from the aneurysm had begun in the thoracic aorta, probably on a syphilitic basis, and had extended to the iliacs. There was in addition another aneurysm of the proximal thoracic aorta above the point at which the dissection had begun beneath an atherosclerotic plaque.

The general principles of the management of dissecting aneurysm of the aorta which have been employed successfully have had in common the restoration of a relatively normal channel of blood flow. This had been accomplished in several ways. The most frequently used approach has been to divide

the thoracic aorta distal to the origin of the left subclavian artery and to suture the intima media and adventitia together around the circumference of the proximal end of the distal segment of the divided aorta. A window is then cut in the intima above so that the aorta proximally still has two channels for blood flow the normal one and the intramural one.⁷ This is referred to as the fenestration procedure. In other instances an aortic homograft has been inserted following excision of a segment of the thoracic aorta. These two methods of technical management are shown in Figure 45.

Prognosis. The benefits of surgery must be evaluated in the light of the extremely serious nature of the underlying disease. Not infrequently the patient will eventually die from further intramural dissection with or without rupture of a proximal aneurysm or from rupture of the dissecting proximal portion of the aorta into the pericardium with pericardial tamponade.¹⁰ Nevertheless the measures outlined above represent the first definite advance in relatively effective management of dissecting aneurysm of the aorta hitherto a disease which has proved fatal in 75 per cent of cases within a very few days. Prompt surgical intervention is thus indicated when the diagnosis is reasonably well established.

Neurologic Complications of Occlusion of the Thoracic Aorta

It was pointed out earlier that perhaps the two major hazards attending occlusion of the thoracic aorta are first, the severe overload that it places upon the heart which may lead to acute heart failure and,

aneurysm. However, at laparotomy it was eventually discovered that the hemorrhage was arising from the celiac axis and, particularly, from the splenic artery. The dissecting aneurysm had dissected out along the splenic artery, rupturing through the adventitia, and this was the site of the hemorrhage. It was assumed that the patient did have a dissecting aneurysm which had arisen probably in the thoracic aorta, but the patient's condition was so poor that it was decided simply to ligate the splenic artery proximal and distal to the point of hemorrhage and to divide it between the ligatures. Since control of the hemorrhage had been accomplished, the abdomen was closed. The patient expired two days later from no truly obvious cause. He did not bleed again, and he had not been oliguric long enough to develop a serious uremic state. The blood pressure was markedly elevated until very shortly before he went into sudden collapse and died. The use of antihypertensive drugs was considered but was postponed, since he had previously been in shock due to blood loss into the abdomen. Autopsy revealed that the dissection from the aneurysm had begun in the thoracic aorta, probably on a syphilitic basis, and had extended to the iliacs. There was in addition another aneurysm of the proximal thoracic aorta above the point at which the dissection had begun beneath an atherosclerotic plaque.

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the subclavian artery and procedures including ligation of the lower thoracic intercostal vessels are likely to cause spinal cord damage. The reason temporary occlusion of the aorta below the subclavian artery is followed by ischemic changes in one individual and not in another is explained by the extreme differences that are possible in the segmental pattern of the spinal cord blood supply.

Even with the use of hypothermia tube shunts or extracorporeal bypass aortic surgery may still cause spinal cord damage when the lower intercostals are occluded as in the resection of an extensive aneurysm of the thoracic aorta. The danger of ischemic cord injury during resection of coarctation of the aorta when the collateral circulation is not well established or in the presence of a patent ductus entering the aorta below the level of the coarctation or when there is a considerable flow of blood through the narrowed but not obliterated channel at the level of the coarctation was mentioned previously. In fact it accounted for 11 of the 24 instances of spinal cord ischemia reported by the above authors. The young individual is somewhat protected against spinal cord damage by his greater resistance to anoxemia and better potentialities of collateral circulation because of relatively larger spinal vessels. The vessels of the elderly person are atherosclerotic and thus there may be narrowing or even occlusion of some of the intercostal or still smaller arteries along the spinal cord itself.

Neurologic complications after aortic surgery may also be caused by ischemia of the peripheral nerves to the lower extremities as well as resulting from prolonged occlusion of the infrarenal aorta. The mech

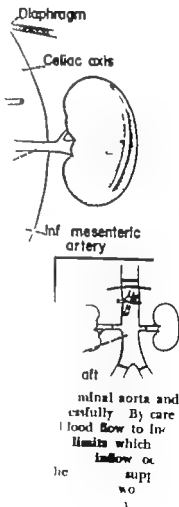
second, the danger of neurologic damage. Occlusion of the aortic arch proximal to the origin of the great vessels which supply the brain cannot be tolerated without the use of a pump oxygenator or a bypass shunt from the proximal ascending aorta to the carotid arteries both to supply the brain with blood and to take the load off the heart. On the other hand, the heart usually can tolerate for a time cross-clamping of the thoracic aorta distal to the origin of the left subclavian artery or even proximal to this point—and of course the brain is well supplied with blood whenever the aorta is clamped distal to the carotid arteries. Therefore the particular hazard of clamping the thoracic aorta distal to the origin of the innominate and left common carotid arteries is that of ischemic damage to the spinal cord. Various time limitations have been suggested by different authors during which total occlusion without bypass around the point of occlusion of the thoracic aorta can be safely imposed. In a recent report it was suggested that 18 minutes or less is probably the safe limit which can be tolerated by most patients¹. On the other hand, occlusion of the thoracic aorta for as long as an hour in the course of resection of aneurysms has been tolerated by some subjects with only mild paresthesias in the legs which cleared up within a week or so. Adams and van Geertruyden¹ reported 24 instances of ischemia of the spinal cord associated with surgery involving the thoracic aorta—11 after operation for coarctation, seven after aortic resection and six after temporary occlusion of the aorta for one reason or another. Their conclusions were as follows: only temporary occlusion of the thoracic aorta below

the subclavian artery and the occlusion of the lower thoracic aorta to cause spinal cord damage. Occlusion of the aorta followed by ischemic damage not in another is explained that are possible in the spinal cord blood supply.

Even with the use of extracorporeal bypass and spinal cord damage was occluded as in the resection of the thoracic aorta. Injury during resection when the collateral circulation or in the presence of aorta below the level of a considerable flow of but not obliterated circulation was mentioned previously for 11 of the 24 instances reported by the above author. It is somewhat protected by its greater resistance to occlusion of collateral circulation. Larger spinal vessels are atherosclerotic and or even occlusion of smaller arteries along the

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anastomoses of the graft to the aorta. In other instances the graft continued through the aorta. The anastomoses were anastomosed and arterial blood was flowing.



anism of this may be similar to that of spinal cord ischemia, but usually there is evidence of peripheral circulatory deficiency as well. Actually, most surgeons have found that the aorta may be clamped below the level of the renal arteries for at least an hour without producing nerve damage except in the rare patient. In many instances, particularly in the presence of ruptured aneurysm of the abdominal aorta, the clamps have been left on the aorta for three or four hours without permanent damage, though this has resulted in temporary weakness in the legs. The period of unrelieved arterial occlusion should always be minimized by careful planning and expeditious surgery.

The Thoraco-abdominal Aorta and Its Visceral Branches

Thoraco-abdominal Aortic Aneurysms The successful resection of thoraco-abdominal aneurysms^{23, 30} with grafting has been a technical feat scarcely less impressive than that of resection of aneurysms of the aortic arch. For in the case of the former the celiac, superior mesenteric, and renal arteries may all have to be dealt with.¹⁸

Much has been learned in recent years regarding the length of time which the arterial supply to the liver, bowel, and kidney arteries can be interrupted without irreversible changes in these essential viscera. Happily, in a limited number of cases aneurysms involving all these vessels have been successfully resected and a graft inserted (Fig. 47). In general the tactic employed has been to use a system of temporary or permanent bypass shunts to afford blood supply

to the viscera while the anastomoses of the graft to be inserted were accomplished. In other instances blood flow has been permitted to continue through the aneurysm while visceral arteries were anastomosed to a prosthesis through which arterial blood was flow

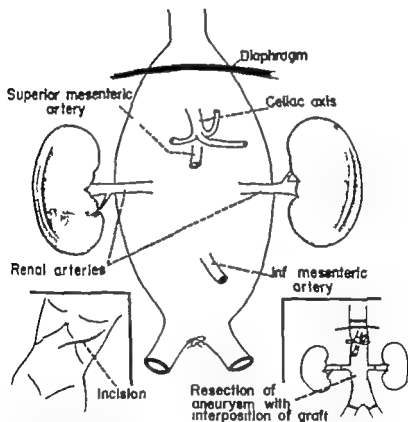


FIG. 47 Aneurysms involving the entire abdominal aorta and its visceral arteries have now been resected successfully. By careful planning the length of time the arterial blood flow to individual organs is occluded can be reduced to limits which are satisfactorily tolerated. The kidneys withstand inflow occlusion less readily than do the bowel and the viscera supplied by the celiac axis. The incision shown in the inset would be extended to the symphysis pubis if the lower portion of the abdominal aorta were found to be involved.

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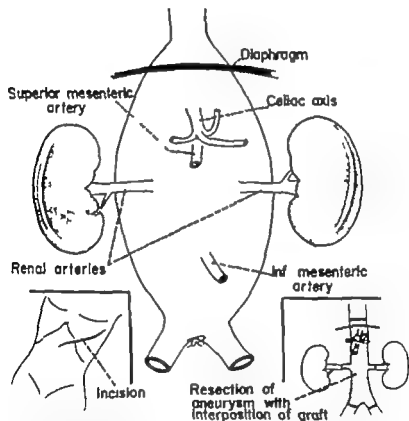


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ing Whereas the procedure might be considered formidable and truly is, it can be performed by painstaking and methodical surgery We resected an aneurysm involving all these vessels in one patient The anastomoses were all successfully completed with the use of an aortic homograft on which the renal arteries, superior mesenteric arteries, and the celiac axis had been left long when the aorta was taken at autopsy In this patient the aneurysm was resected in stages, blood flow to individual viscera being interrupted for only a few minutes as the anastomoses of the graft to visceral arteries were successively performed Unfortunately the patient died in the postoperative period, apparently from cardiac failure The procedure had taken almost nine hours, and a large volume of blood loss had been replaced However, on the basis of the experience gained with this case we shall unquestionably shorten the operating time in the future, and certainly the blood loss can be reduced

DeBakey, Creech and Morris²³ reported resection of four such extensive thoraco-abdominal aneurysms of the aorta In all of these cases the aneurysm extended from the lower descending thoracic aorta to the lower abdominal aorta and involved the celiac, superior mesenteric, and one or both renal arteries, as was the situation in our case mentioned above They emphasized that the most important consideration in the extirpation of this type of aneurysm is the possibility of ischemic damage to such vital organs as the kidney, liver and gastro-intestinal tract as a consequence of temporary arrest of circulation to these organs during resection of the aneurysm Hypothermia has been employed by some surgeons, but

the use of various types of temporary or permanent bypass shunts is preferred. In our own experience as in that of DeBakey and his associates, the use of hypothermia has presented certain problems which can be avoided by the use of bypass shunts for preserving adequate blood flow to such structures as the brain spinal cord and the essential abdominal viscera. Three of the four cases reported by DeBakey and his associates lived. The period of occlusion of the renal arteries ranged from 15 to 46 minutes. Serial renal function studies in these cases revealed a characteristic pattern of depression of function as reflected in an increase in the blood urea nitrogen level and a significant reduction in renal blood flow and glomerular filtration rate during the first four or five days after operation with a progressive return to normal during the subsequent ten days to two weeks. The period of occlusion of the celiac artery in these cases ranged from 44 to 116 minutes but studies of hepatic function revealed no significant alterations. The period of arrest of the circulation through the superior mesenteric artery varied from 36 to 102 minutes and no significant disturbances in gastro-intestinal function were observed. Subsequently Lillehei and his associates²⁴ demonstrated that in dogs the intestine can be successfully homografted and that frequently the period of occlusion of the vascular flow through the intestine to be autografted or homografted can be extended to many hours if the intestine is cooled. In other words the tolerance of the liver and the bowel for inflow arterial occlusion is considerably greater than had previously been realized. Lillehei and his associates found that the majority of adult

mongrel dogs tolerated up to 3.5 hours of superior mesenteric arterial occlusion if the collateral circulation to the small bowel was not disturbed. If all collateral circulation to the bowel was occluded as well, most dogs died following a 3 to 3.5 hour occlusion of the superior mesenteric artery. Interruption of all circulation through the small bowel for two hours was tolerated if the bowel was first allowed to cool to room temperature. If the bowel was cooled to 5° C, then it was safe to interrupt all circulation to the small bowel for at least five hours, with subsequent survival of the bowel. No external cooling of the bowel removed from the animal was required if it was returned to the peritoneal cavity and circulation restored within two hours. An interesting by-product of these studies was the observation that division of all connections with the central nervous and lymphatic systems was tolerated. It was found that lymphatic connections regenerated within a period of days.

It is apparent that the abdominal viscera can usually survive temporary periods of occlusion of blood flow up to one hour with the partial exception of the kidney. Actually, it has been pointed out that permanent interruption of the celiac blood flow can be survived and that at times even acute interruption of blood flow through the superior mesenteric artery by an embolus from the heart may be survived because of the collateral blood flow from the superior pancreaticoduodenal artery arising from a branch of the celiac axis and from blood flow received through the inferior mesenteric artery, as well as from the retroperitoneal collateral arteries. With respect to renal

arterial flow by suitable planning one can in most instances maintain blood flow to one renal artery while the anastomosis of the graft to the other renal artery is being accomplished. Thus one can often avoid complete arterial inflow occlusion to both kidneys simultaneously. Even so if complete occlusion of both renal arteries were required as a temporary expedient because of the exigencies of the technical situation the blood flow could be re-established to one kidney within 15 to 20 minutes and this period of occlusion has been found to be tolerated without irreversible changes in most patients.

Aneurysms of Abdominal Aorta Visceral Branches

The Celiac Axis and Superior Mesenteric Artery Aneurysms involving the celiac axis^{11 44 47} and the superior mesenteric artery⁴⁸ have been managed successfully. In general these cases were treated either by actual excision of the aneurysm with ligation of the artery distally and proximally or by aneurysmorrhaphy. Undoubtedly excisional therapy with by pass graft will be substituted for simple ligation and excision in the future. Perhaps this has been done already but such reports have not come to our attention. Despite the serious risk of hepatic or intestinal ischemia following excision of the patent aneurysm involving the major blood flow to these viscera it has been pointed out above that in some cases this will be tolerated. Aneurysms of the hepatic artery have led to bleeding into the common bile duct and have been a source of massive hemorrhage into the alimentary tract⁴⁹.

Aneurysms of the Renal Artery Aneurysms involving the renal artery were considered in discussing aneurysms of the thoraco-abdominal aorta. The renal arteries are involved from time to time either by isolated aneurysms, perhaps in association with post-stenotic dilatation in a case of renal ischemia due to renal arterial occlusive disease, or in association with a generalized aneurysmal enlargement of that portion of the aorta from which they arise. In general renal blood flow must be restored by means of a graft following resection of the aneurysm. Again, as has been emphasized in discussing the various diseases of the aorta, the surgeon must be mentally prepared to deal with whatever specific variation in the underlying basic diseases of occlusion or aneurysm formation is discovered at the time of surgery. It scarcely need be mentioned that a wide acquaintance with the available literature on the subject is of much assistance to the operator.

The Infra renal Aorta and the Ilac Arteries

A vast experience has been accumulated over the past ten years in the diagnosis and technical management of aneurysms in the lower abdominal aorta, the most frequent site of such lesions ^{4, 10, 20, 22, 31, 80, 90}. Hitherto an almost hopeless situation, the successful excision and grafting of these aneurysms, first accomplished by Dubost and his associates ^{25, 26} in 1951, is now a routine operation in most major hospitals.

The *prognosis* of the patient with an abdominal aortic aneurysm arising below the renal arteries does deserve comment. There are still many patients and physicians who temporize with aneurysms until they

rupture which immediately greatly reduces the chances of successful resection of the leaking aneurysm and the insertion of a prosthesis. The poor prognosis which is associated with abdominal aortic aneurysms was documented earlier in this discussion.

There is rarely valid excuse for temporizing with aneurysms of the abdominal aorta that are of significant size or that are symptomatic.

Whether or not an aneurysm is palpable through the abdominal wall depends upon the size of the aneurysm, the degree of obesity of the patient and whether or not the aneurysm is filled with clot and thus does not present an expansile pulsation. An aneurysm of the abdominal aorta is not infrequently associated with a separate one in the thoracic aorta as well.⁷³

Diagnosis

The diagnosis of an abdominal aneurysm can be exceedingly easy or it can be most difficult. In the thin individual the appearance of a pulsating mass in the epigastrium or in the region of the umbilicus will commonly be noticed even by the patient himself. This usually is discovered long before the aneurysm has enlarged to the point of producing pain or serious threat of rupture. There is a common misunderstanding that the aorta bifurcates at about the level of the symphysis pubis which is of course erroneous. The bifurcation of the aorta is at approximately the level of the umbilicus. Therefore when one palpates an aneurysm at this level it is situated at the termination of the aorta and may and frequently does involve one or both iliac arteries. Just as the diagnosis of an aneurysm is made earlier in the

thin individual, so is resection of the aneurysm with the insertion of a synthetic prosthesis far more easily accomplished in the thin than in the obese subject.

In contrast to the ease with which the infrarenal aortic aneurysm can usually be diagnosed in the thin person, the aortic aneurysm in the obese subject may actually go on to the point of rupture prior to its discovery. During recent years we have operated upon obese patients for ruptured aneurysm who had been explored previously for abdominal pain interpreted as being due to intestinal obstruction. In other instances the patient was thought to have a renal or ureteral calculus, since leakage from the aneurysm had resulted in hematoma formation in the left retroperitoneal space which caused pain to radiate from the left flank into the genital region on that side. In still other instances the patient has been considered to have arthritis of the spine, with or without radiologically apparent erosion of the spine. It is particularly difficult to arrive at the diagnosis of erosion of the spine by an aneurysm when there is no calcification in the wall of the aneurysm and when there is demineralization of the spine not only in the lumbar area but also in the thoracic area. At other times we have unexpectedly encountered an aneurysm in an obese person when the abdominal exploration had been performed for ischemic changes in the legs, complete occlusion of the aneurysm by thrombus had occurred and the patient presented the findings of Leriche's syndrome. Aortic aneurysm may rupture into the alimentary tract, or into the vena cava, or produce retroperitoneal hemorrhage in which infection may develop and cause fever.³¹

In summary any patient with abdominal pain of undetermined or indeterminate origin should be considered as possibly having either partial occlusion of one or the other of the visceral branches of the aorta or an aneurysm.

Radiologic Findings.¹⁸ When an aneurysm of the abdominal aorta is suspected in the majority of cases it can be demonstrated with plain P A and lateral films of the abdomen (Fig 41). The aneurysm which may or may not be palpable in the obese subject is visualized because its wall is often calcified. Frequently this is not particularly apparent on a P A

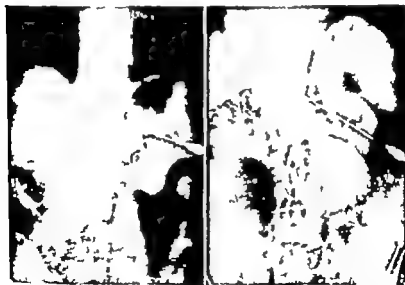


FIG. 48 Aortogram of a normal and of an aneurysmal lower abdominal aorta. At the left is shown a normal aortogram of the lower abdominal aorta performed with a limited amount of contrast medium for the purpose of excluding renal arterial occlusive disease in a hypertensive young man. The poor visualization of the iliac vessels was due to inadequate filling. On the right is shown an aortogram which discloses a fusiform aneurysm.

film, but it is often readily visualized on the lateral film. This calcification is not always present, however, and at times one will wish to perform an aortogram (Fig 48) to exclude aneurysm where the presence of such a lesion is uncertain. In this way the patient may be spared an unnecessary laparotomy. On the other hand, if one can palpate an aneurysm in the lower portion of the aorta and especially if the calcification in the wall of the aorta shows dilatation on

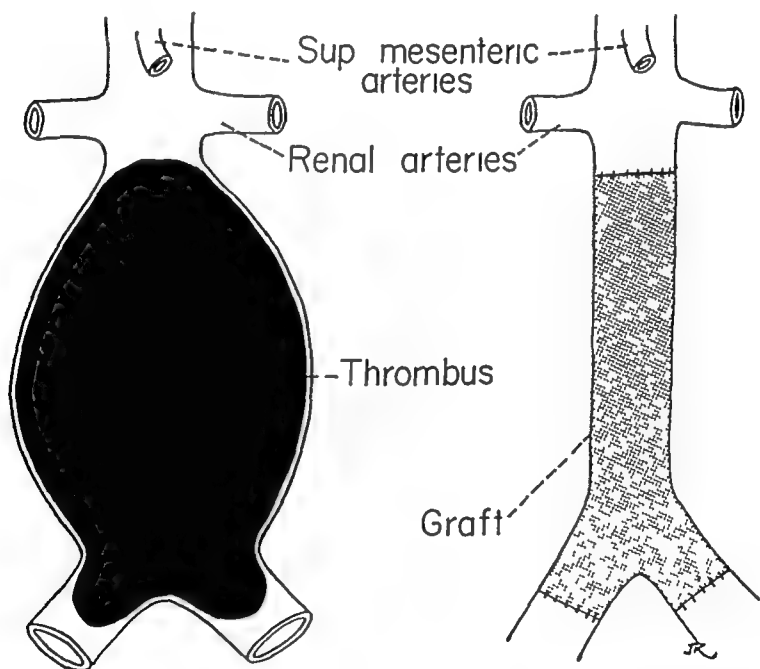


FIG 49 The typical fusiform aneurysm of the infrarenal aorta contains thrombus, but it is not often completely occluded and femoral pulses are usually present. Occasionally, however, the patient presents himself with the signs and symptoms of lower aortic occlusive disease ("Leriche syndrome"), and in the relatively obese subject the completely thrombosed aneurysm may not even be suspected until the abdomen has been opened—unless calcification in the wall of the aneurysm was visualized preoperatively on roentgen examination.



FIG. 50 Aneurysm of infrarenal aorta. This typical aneurysm of the lower abdominal aorta was resected and a graft prosthesis interposed. Such operations are routine in most of the larger hospitals and the results are satisfactory.

the plain roentgen studies, we prefer to operate without performing an aortogram, in most instances. In the occasional case where there is occlusion of the lower aorta with evidence of Leriche's syndrome (Fig 49), we perform an aortogram for the primary purpose of determining whether or not there is a sufficient run-off in the iliac and femoral vessels to afford an opportunity for successful bypass grafting if this is indicated in the given case (Fig 50).

In addition to the calcification in the wall of the aneurysm and the visualization of the aneurysm on aortogram, additional findings which may be present in one case or another are erosion of the spine by the aneurysm (best seen in the lateral films of the spine), displacement of various structures such as the kidney or portions of the alimentary tract which can be visualized on barium study, and by a soft tissue mass seen in the abdomen on the plain film.

Management

Unruptured Infrarenal Aortic Aneurysms It is preferable to consider the treatment of unruptured and ruptured abdominal aortic aneurysms separately, though the principles of surgical management are basically the same in each instance. In addition to the fact that the technical procedures are easier when the aneurysm is not ruptured, the prognosis is vastly better in the patient whose aneurysm has not ruptured. This is due to a variety of factors, the principal one being that the patient whose aneurysm has ruptured acutely usually is admitted in shock, and one is confronted with all the hazards of such circumstances. At operation it is often hard to identify the

iliac arteries the aorta proximal to the point of rupture the iliac veins the vena cava and the ureters because of the massive retroperitoneal hematoma—and meanwhile blood loss continues until the proximal aorta and the iliac arteries have been controlled.

The optimal approach to the abdominal aorta is through a long midline incision which extends from a point shortly below the xyphoid process to a point well below the umbilicus (Figs 51A, 51B, 51C). This gives access to the aorta below the renal arteries but proximal to the beginning of the aneurysm above

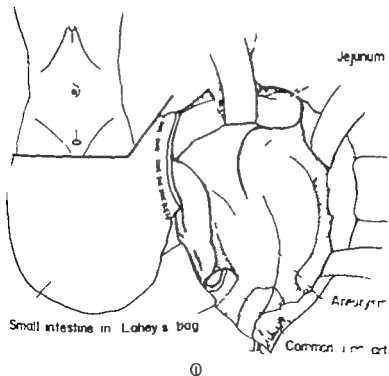


FIG. 51A. Steps in resection of infrarenal aneurysm of abdominal aorta. (1) Long midline incision and isolation of small intestine in a Lohey bag.

and it affords adequate visualization of the common iliac arteries distal to the aneurysm. When the aneurysm involves the common iliac arteries one will need to secure control of the external iliac arteries and, as soon as feasible, the internal iliac arteries. In our experience the major hazard of serious bleeding encountered in the resection of an

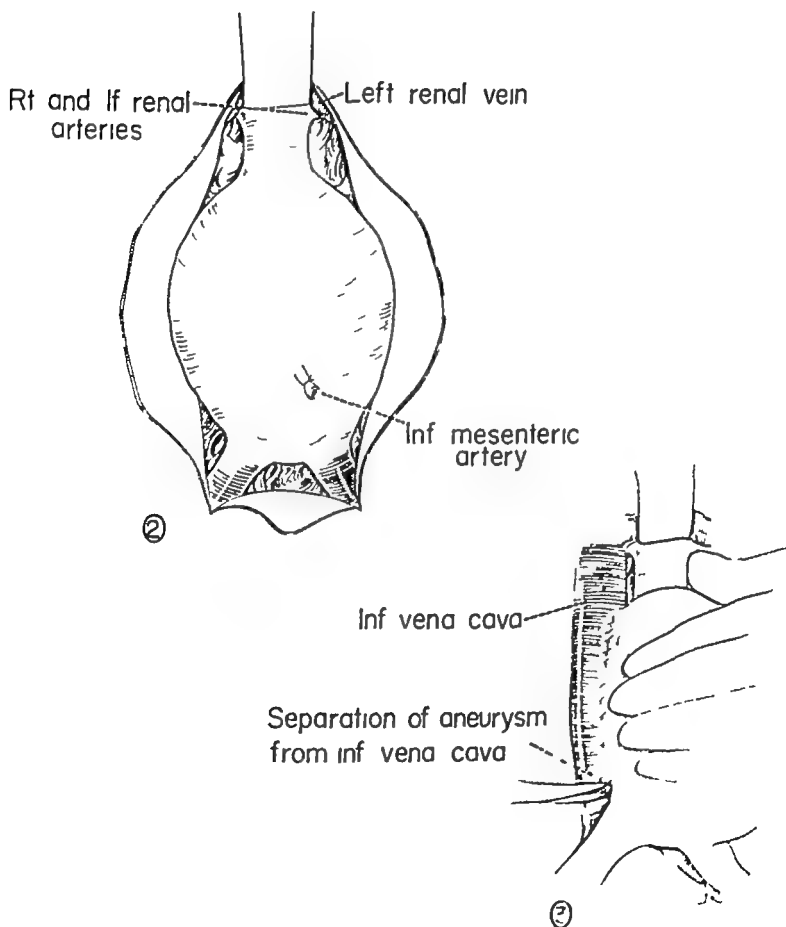


FIG 51b (2) Exposure of infrarenal aorta above aneurysm, and ligation and division of inferior mesenteric artery (3) Limited dissection to permit safe passage of clamp anterior to the inferior vena cava but posterior to the aneurysm

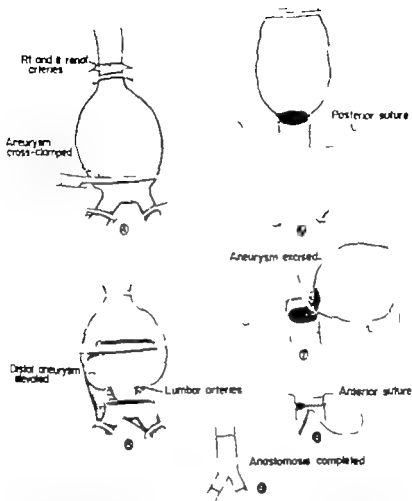


FIG. 31c (1) The aneurysm and the common iliac artery have been cross-clamped (5) The distal end of the aneurysm has been turned upward to permit visualization of lumbar artery and the inferior vena cava. A portion of the outer wall of the aneurysm is left on the vena cava where necessary to avoid injury (6) The aneurysm has been elevated from its bed proximally to the point selected for anastomosis to the normal aorta. The posterior row of sutures may be placed while the aneurysm is still attached anteriorly. This affords an additional margin of safety in certain instances (7) The aneurysm is completely excised (8) The anterior suture line is placed (9) The bifurcation prosthesis has been anastomosed to the common iliac arteries below

aneurysm of the aorta below the renal arteries is occasioned by injury to an iliac vein or to the inferior vena cava. Injury to the iliac veins is particularly likely to occur as one is gaining control of the iliac arteries just adjacent to the terminal portion of the aorta. The junction of the iliac veins is situated immediately behind the bifurcation of the lower aorta and, should one injure these veins prior to getting exposure through mobilization of the aorta at this point, suture of the laceration is difficult. Nevertheless, probably few patients have been lost because of bleeding from these large veins, certainly we have lost none, nevertheless, it does prolong the operation and it requires the transfusion of more blood than would otherwise have been necessary. The vena cava itself is particularly likely to be injured just as it emerges from beneath the aorta or, more accurately, alongside the aorta at the right of the aortic bifurcation. Here again, this can be avoided by dissection into the wall of the aneurysm, leaving a portion of the adventitia of the aneurysm adhered to the vena cava, at least until the aneurysm has been removed. Once the mass of the aneurysm has been resected, one can dissect in the region of the vena cava with great facility and precision, and injury is rare. Furthermore, even should injury occur at this time the repair of the small rent in the vena cava is a simple matter.

It is not usually difficult to gain control of the aorta proximal to the aneurysm but below the renal vessels when there is as much as a 25 cm. distance between the origin of the right renal artery and the beginning of the aneurysm. If there is a clot in the aneurysm

which extends up to the level of the renal arteries it may be necessary temporarily to occlude the aorta just above these vessels and to perform an endarterectomy or at least to flush out the thrombus from the segment of normal aorta just below the renal arteries a second clamp then being placed below the renal arteries and the one above the renal arteries is removed. When there is thrombus in the aneurysm however one should check the pulsations in the renal arteries after completion of the proximal anastomosis to be certain that neither of these vessels has been occluded with particles of thrombus dislodged from the aorta.

Once one has gained control of the common iliac arteries below and of the aorta above the aneurysm usually with a Crafoord or Satinsky clamp the situation is well in hand. Some surgeons still prefer to divide all possible lumbar arteries and to dissect the aneurysm away from the vena cava prior to closure of the clamps above and below and actual excision of the aneurysm itself. This does minimize aortic occlusion time. However we ourselves following the example of DeBakey and his associates have found that it is far simpler boldly to enter the aneurysm following occlusion of the aorta above and the common iliac arteries below and to carry the dissection forward from within the aneurysm after evacuation of the clot which is almost always present to some degree. In this way one can resect most if not all of the aneurysm without injuring the vena cava. If there is excessive bleeding from intercostal vessels, the aneurysm can be essentially transected and each end cross-clamped with long Kocher clamps. Thus bleeding is allowed to occur into the halves of the aneurysm but blood is not

lost extravascularly and in this way bleeding is minimized. It is surprising how great is the variation in the bleeding from the lumbar arteries in different patients. In some subjects it is not necessary to attempt to control the bleeding into the aneurysm from the lumbar arteries by either suitably placed clamps or by compression with packs, since the bleeding from these vessels is very slight indeed. Of course, as one retracts the portions of the aneurysm anteriorly he visualizes the lumbar arteries beneath and clamps each pair progressively from below upward to the point at which the aorta is to be divided at the most proximal extension of the aneurysm. The proximal segment of the aneurysm is usually dealt with first, it is freed up, transected at its origin and the proximal anastomosis of the transected proximal aorta to the synthetic prosthesis (Teflon or Dacron) is completed. We formerly injected heparin into iliac arteries distally, but in the last two years we have abandoned this practice. There has been no instance of clotting in the arteries to the legs during the period of occlusion, except for clotting in the iliacs just distal to the clamp. This blood is invariably aspirated with the suction tip just prior to the completion of the lower anastomosis. In fact, it is well to place bulldog clamps across the iliac vessels just above the level of the internal iliac or hypogastric arteries, and to remove all blood from the common iliac vessels proximal to this level. No blood clot must be allowed to remain in the prosthesis, or in the proximal aorta, or in the common iliac arteries at the time of completion of the distal anastomosis and the restoration of normal blood flow to the lower extremities. Otherwise embolization to the arteries in the legs may occur.

The mortality rate following resection of unruptured aneurysms of the lower abdominal aorta can be less than 5 per cent in patients in otherwise good physical condition. There has been considerable interest in the effect of cross-clamping the infrarenal aorta on renal function²⁴ but we have noted no serious ill effects from this in patients with previously normal renal function. Severe intestinal ileus can occasion much morbidity and for this reason a long intestinal tube is inserted routinely preoperatively. The common causes of postoperative deaths in such patients in our experience have been coronary occlusion and hemiplegia. Late results^{25, 26} and certain complications²⁷ have been published.

Occasionally the iliac arteries are involved and a Y-graft must be employed. If the iliac arteries are found to be thrombosed the Y prosthesis must extend from just below the renal arteries to the common femoral artery on either side.

Ruptured Abdominal Aneurysms.^{28-36 47 50 51} Much has been learned regarding the natural history and course of the aneurysms of the lower abdominal aorta in recent years as reviewed above. In particular abdominal pain frequently is present for some time prior to rupture of an aneurysm and aneurysms which are symptomatic should be operated upon immediately. However in the obese subject in whom the cause of the pain has not been identified rupture of the aneurysm following its gradual enlargement can occur without signs which were considered significant to the observers. Moreover the abdominal or back pain which the patient has may not be due to the aneurysm; in one patient the back pain proved to be due to lumbar spine metastases from prostatic carcinoma.

An aneurysm which is leaking may manifest itself in a variety of ways. First, it should by no means be thought that the rupture of an aneurysm of the aorta is invariably fatal immediately, in the absence of resection. As a matter of fact, many ruptured abdominal aneurysms give pathologic evidence of having leaked at several intervals over past months. We successfully operated upon one patient who had a ruptured abdominal aneurysm that had manifested itself as possible intestinal obstruction for which the patient has previously had two abdominal explorations which were recorded as being negative. Unquestionably the pain in each instance was due to a rupture of the aneurysm, with sealing off of the point of leakage. Perhaps in even a majority of patients the symptoms of beginning rupture have been present for several days or at least many hours prior to the time at which the correct diagnosis is made, whether or not surgical intervention is attempted. Actually it should always be attempted if the patient is not moribund, for if the subject survives operation he may also survive oliguria due to the period of hypotension and perhaps suprarenal aortic occlusion. The fact that the patient suddenly exhibits abdominal pain, with a drop in the hemoglobin and hematocrit levels but without evidence of bleeding into the chest or into the alimentary tract, should certainly suggest the possibility of ruptured aneurysm. The signs and symptoms of acute rupture usually are abdominal, flank or back pain associated with an enlarging abdominal mass which frequently pulsates. The concomitant decline in the hemoglobin and hematocrit levels have been mentioned.

In the cases of ruptured aortic aneurysms that we have operated upon an aortogram was not performed because the diagnosis was obvious. However the diagnosis was in error in two patients who had masses in the left flank and lowered hematocrit levels: one subject had a highly vascular retroperitoneal sarcoma which had been the site of hemorrhage and the other had a massive retroperitoneal hematoma which developed secondary to long acting heparin therapy which he had been receiving for a cerebrovascular accident. Furthermore in two instances of ruptured abdominal aneurysm the patient had been in our hospital for one week and three weeks respectively. The first patient had been admitted to the urology service with the diagnosis of left ureteral colic, since he was obese and had pain radiating from the left flank and into the scrotum. Five days following admission during which period two successive drops in the hematocrit level had been observed a pulsating mass suddenly was palpable in the lower abdomen. He was taken immediately to the operating room and an aneurysm of the lower abdominal aorta and left iliac artery was successfully resected and a homograft inserted. In the instance of the other patient he was studied for obscure abdominal pain for a week or so on the diagnostic service prior to the abrupt further bleeding from the aneurysm. By the time the surgical team had been called he was virtually moribund and he succumbed on the operating table as the abdomen was being opened. Resuscitation was unsuccessful.

These various comments are made with the specific purpose of urging the prompt suspicion of the presence of a ruptured aneurysm and exploration of the

abdomen whenever this diagnosis is a reasonably good possibility. Usually the films of the abdomen which disclose calcification in the aneurysm, the presence of an expanding mass in the abdomen (usually on the left) and a fall in the hematocrit level will suggest the diagnosis of ruptured aneurysm.

The chief barrier to successful management of a ruptured aneurysm lies in the fact that one begins the operation with a patient who is losing massive quantities of blood into the abdomen, and the outcome here is very likely to be what it is when one operates upon any other patient in similar circumstances, regardless of whether or not the operation is being performed for aneurysm. That is, the general condition of these patients is extremely poor and, should one manage to control the aortic leak and then to continue the restoration of an adequate blood volume, the complications of coronary occlusion, hemiplegia, or postoperative renal failure are always going to take their toll. It is relatively meaningless to discuss the mortality rate, since this figure will depend upon widely varying factors including the state of the patient when he reaches the hospital and the nature of the aneurysm after one has opened the abdomen. We have successfully resected and grafted ruptured aneurysms only to have the patient die two weeks later of postoperative renal insufficiency, due to the prolonged shock he sustained before and during the early part of the operation. This is not to say, however, that one should not always try to resect the aneurysm and put in a graft, since in a number of cases this will be successful, and there is no sure way of determining in advance which patient will live and which patient will not. They will virtually all die without operation.

Aneurysms of the External Iliac, Femoral and Popliteal Arteries

Aneurysms occur in all arteries at all levels though with different frequency. Aneurysms of the common iliac arteries have been considered in connection with aneurysms of the lower aorta since aneurysms of the lower aorta frequently involve one or both of the common iliac arteries. Aneurysms of the external iliac artery have been relatively uncommon in our experience. We have had a number of aneurysms which have involved the femoral or popliteal arteries (Fig 52). Such aneurysms may have a dissecting component.²³

In contrast to the problems associated with the management of aneurysms of the aorta and the common iliac arteries the management of aneurysms of the arteries of the extremity is essentially a simple matter. One first gains control of the artery both above and below the level of the aneurysm and he then carefully separates the concurrent vein from the aneurysm, whether it be the femoral vein or the popliteal vein. Important nerves must also be identified and retracted to avoid harm. The identification of artery, vein and nerve is best achieved by carrying the incision proximal to a point where the tissue planes have not been obscured by inflammatory reaction around the aneurysm itself. Having passed tapes around the vein, nerve and artery where these various structures exist together one can then carry the dissection downward under direct vision and the three can be carefully preserved. This identical approach is used to gain access to the aneurysm at a point below its most distal extension.



FIG 52 Ruptured atherosclerotic aneurysm of popliteal artery. This patient had an atherosclerotic aneurysm of the popliteal artery which had ruptured to produce a pulsating hematoma. In the upper photograph the aneurysm may be seen beneath the retracted sciatic nerve and its branches. In the lower photograph the tip of the forceps indicates the point

The anatomic exposure of the external iliac and the femoral artery offers no problems. In contrast the popliteal artery can be difficult to expose.^{8, 12} It may be approached either with the patient in a face-down position or with a medial incision beginning just above the popliteal space and extending to a point slightly beyond this level. The sciatic nerve overlies the popliteal aneurysm usually and thus must be carefully preserved (Fig. 52). Aneurysms of the popliteal artery²² are usually due to atherosclerosis but they may also be due to syphilis or other causes.

One point of importance which should be stressed is the frequency with which aneurysms of the extremity may give rise to signs and symptoms of an infection.²³ To incise an aneurysm without prior preparation for proximal and distal control of the main artery or without adequate blood for replacement if necessary can prove awkward indeed.

Summary and Conclusion

1. The types, etiology, clinical findings, diagnosis and surgical treatment of representative arterial aneurysms have been reviewed. True aneurysms of the aorta are usually due to atherosclerosis or less commonly syphilis. Infection and medial necrosis are etiologic factors in some instances as is trauma.

2. The majority of patients with a symptomatic aortic aneurysm whether thoracic or abdominal will be dead within six months unless the lesion is successfully treated surgically.

at which primary anastomosis of the popliteal artery was performed following resection of the aneurysm. No graft was required in this case since only a short segment of the popliteal artery was involved by the aneurysm.

3 The diagnosis of these lesions rests upon the history and physical examination, plain roentgen films which disclose a mass in the thorax or calcium in the wall of an abdominal aneurysm, fluoroscopy which discloses expansile pulsations of a thoracic lesion, and appropriate angiography where indicated

4 Abdominal pain of undetermined origin may be due to an aneurysm. The discomfort may radiate to the back, often due in part to erosion of the lumbar vertebrae, or it may extend into the scrotum if rupture of the aneurysm has occurred

5 Aneurysms of all segments of the aorta and its major branches have now been successfully operated upon. Some lesions can be excised using lateral aortorrhaphy but most must be resected and grafted

6 Defects in the aortic arch and innominate and carotid arteries must be approached in such a way that brain ischemia is avoided. An additional consideration in the case of aneurysms of the ascending aorta and aortic arch is that of preventing excessive back pressure upon the heart, causing failure of this organ. This objective is best accomplished with the use of the pump oxygenator, in the case of an aneurysm of the ascending aorta, or the use of a system of temporary or permanent bypass shunts when the arch but not the ascending aorta is involved. Similar techniques using temporary bypass grafts are used to resect aneurysms of the descending thoracic and thoracoabdominal aorta. Aneurysms of the infrarenal portion of the aorta can be safely excluded, excised and a graft inserted without serious hazard of distal ischemic changes

7. The patient with a dissecting aneurysm of the

thoracic aorta a condition fatal within a few months in perhaps 75 to 90 per cent of cases can now be offered a fairly effective palliative operation

8 Ruptured abdominal aneurysms are not necessarily fatal immediately and many patients with this condition can be saved by prompt operation. Nevertheless the surgical prognosis after a given aneurysm has ruptured is often less favorable than it would have been prior to rupture

9 The surgical treatment of arterial aneurysms is now so gratifyingly effective that few contraindications to prompt operation for these hazardous lesions exist.

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CHAPTER 6

Embolism

ARTERIAL embolism represents a serious and only partially solved problem in medical practice. The increased attention accorded arterial lesions in general in recent years has resulted in a more acute awareness of the frequency with which embolic phenomena are found if they are carefully sought for. Certainly the increasing age of the population renders large numbers of persons susceptible to the heart conditions which provide the greatest number of arterial emboli. Whereas in many instances these emboli are small and produce minimal symptoms few clinical circumstances provide more dramatic findings than does the abrupt occlusion of a major artery such as the bifurcation of the aorta by an embolus usually from the left side of the heart. When such an episode does occur the patient frequently loses one or both legs and not infrequently his life unless successful surgery is possible. Unfortunately many patients with acute arterial embolism are managed conservatively when an embolectomy under local anesthesia might restore blood flow to the part concerned and still other patients are subjected to major operative procedures where less extensive procedures might have sufficed with a greater salvage of life. Yet even if the embolus is successfully

removed, serious underlying heart disease often results in early death of the patient

The purpose of Chapter Six is to review the general problem of arterial embolism, and to indicate the circumstances under which one feels that certain methods of management are most likely to prove effective. The topics to be considered will be discussed under the following headings:

- A Types and Sources of Arterial Emboli
- B Sites of Lodgement of Arterial Emboli
- C Pathophysiology and Mechanisms of Ischemia Due to Arterial Emboli
- D Diagnosis of Arterial Embolism
- E Management of Arterial Emboli
 - 1 Conservative Measures
 - 2 Embolectomy
 - 3 Long-term Prophylaxis Against Recurrence
- F Prognosis in Arterial Embolism
- G Considerations in the Management of Embolism to Specific Arteries
 - 1 Internal Carotid Arteries
 - 2 Arteries to the Upper Extremity
 - 3 Superior Mesenteric and Renal Arteries
 - 4 Aortic Bifurcation
 - 5 Femoral Bifurcation
 - 6 Popliteal Artery

TYPES AND SOURCES OF ARTERIAL EMBOLI

Any material entering the arterial stream can theoretically embolize and thus emboli may consist of air, fat, tumor tissues from heart or lung,¹¹ bullets^{10, 13, 16, 61, 64, 83, 84, 126, 140, 141, 143, 147, 172} and calcified

plaques^{41 172, 188} however most emboli represent dislodged portions of thrombi usually arising in the left side of the heart (Fig 53). Projectiles may also migrate toward the heart in veins.¹⁸² A further rare source of systemic arterial emboli is the venous embolus which has entered the left side of the heart through a patent atrial or ventricular septal defect (paradoxical embolism). We had one such patient ourselves who exhibited multiple paradoxical renal

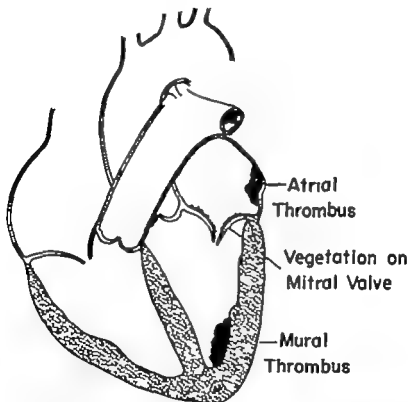


FIG 53 The overwhelming majority of arterial emboli arise in the left side of the heart. Either atrial fibrillation or recent myocardial infarction is found to be present in most instances.

and other infarcts secondary to pelvic thrombophlebitis with arterial embolism due to atrial septal defect (Fig 54) Embolization may also occur distal to thrombosis in the subclavian artery caused by cervical rib compression, or by thrombosis arising in connection with a saccular or dissecting aneurysm¹⁴⁰ of the aorta Thrombosis may also be occasioned by

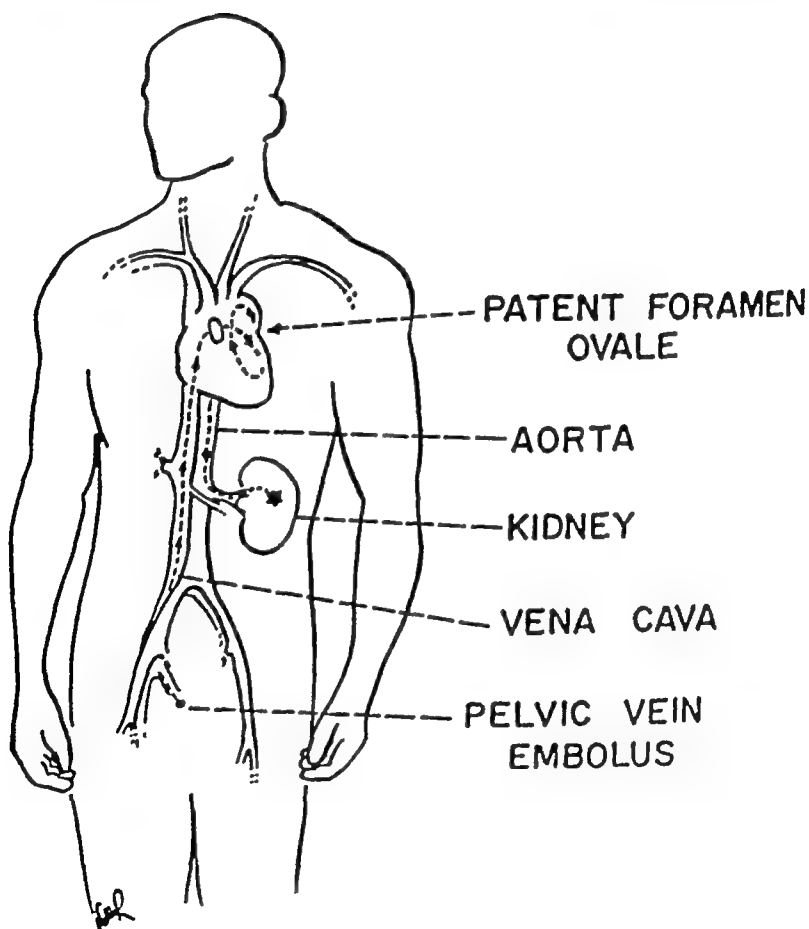


FIG. 54 In the rare case emboli arising in systemic veins find their way into the aorta through atrial or ventricular septal defects

injury to the wall of an artery in the course of surgery or otherwise,¹⁶⁶ and this thrombus may give rise to emboli. Portions of thrombi formed in the pulmonary veins may also migrate into the left atrium and pass as emboli to various parts of the body and thrombus dislodged during aortic surgery¹⁶⁷ may embolize to more distal portions of the arterial bed.

Only emboli secondary to thrombus formation will be considered subsequently.

Again the overwhelming majority of emboli arise in the left side of the heart. Haimovici¹⁶⁸ found that 96 per cent of 330 unselected cases of embolism of the extremities occurring in 228 patients arose from the left chambers of the heart or on the mitral valve. A similar incidence was reported by Warren, Linton and Scannell.¹⁷⁷ Thus the clinical implications are clear, namely that the patients with heart disease are those who are particularly likely to exhibit embolic phenomena and conversely, that the patient who has a clinical picture of acute arterial embolism will usually have heart disease due to myocardial infarction, atrial fibrillation¹⁸⁰ due to one cause or another or subacute bacterial endocarditis with vegetations and thrombus formation on the heart valves.

With respect to the relative frequency of emboli caused by the major forms of heart disease, it is worth noting that in most published series^{88, 100, 176, 177} the fibrillating left atrium has been the source of almost half of the total instances of embolization; next in importance is myocardial infarction with mural thrombosis, accounting for approximately one third of all emboli. The third most frequent cause of thrombus formation and embolization, subacute bac-

terial endocarditis,¹⁷⁴ is of much less importance than the first two given, since this disease produces only about 5 per cent of all emboli. The remaining emboli arise from the various causes listed previously. More recently, intracardiac surgery with thrombosis around sutures or synthetic materials inserted to close septal defects or for other purposes has been noted to produce emboli with some frequency.

SITES OF LODGEMENT OF ARTERIAL EMBOLI

The emboli arising in the left heart and elsewhere may of course enter virtually any artery.^{100, 136, 166} They may involve the coronary arteries^{115, 178} (one case ourselves), retinal vessels, cerebral vessels, those to the upper extremity, visceral arteries within the abdomen, the aortic bifurcation, iliac arteries, femoral

TABLE 3 *Arterial Embolism, 1937-1953*
Emboic Sites—337 Emboli

Site	Emboli	
Internal Carotid and branches	50	14.6%
Upper extremity	59	17.5%
Mesenteric	17	5.0%
Aorta	30	9.0%
Iliac	30	9.0%
Femoral	80	23.7%
Popliteal	33	9.8%
Tibial	8	2.4%
Other*	30	9.0%

(From Warren R., Linton R. R., and Scannell J. C.: Arterial embolism; recent progress. *Ann. Surg.*, 140:311, 1954.)

* Renal, splenic, retinal, skin.

arteries popliteal arteries and more distal portions of the arterial bed (Table 3). Thus the possibility of embolism should be considered in any patient who

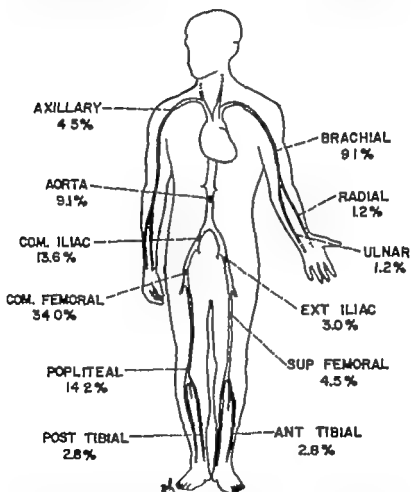


FIG 55. The frequency of arrest of 330 *peripheral* emboli in different arteries is shown (Haimovici H. *Angiology* 1:20, 1950). Most emboli become arrested at a bifurcation of the vessel concerned. The common femoral artery is the vessel most frequently involved by emboli and femoral embolectomy is readily performed under local anesthesia.

TABLE 4. Sites of Peripheral Arterial Emboli—Collective Data

Authors	No Cases	Upper Extremity	Aortic	Iliac	Femoral	Popliteal
Andrus	71	13	10	5	20	23
Austin	24	3	0	2	10	9
Daley	133	39	23	21	29	21
Danzis	127	29	11	23	52	12
Des Prez	82	11	11	10	27	23
Dye	65	14	5	7	23	16
Flisher	212	—	23	20	123	46
Hannovici	312	53	30	55	127	47
Hopkins	45	13	7	2	18	5
Key	380	16	17	66	208	43
Lovingsgood	6	—	4	—	2	—
Missell	11	—	1	3	5	5
McCarthy	86	16	5	13	36	16
Peterson	279	46	31	48	126	28
Prompt	13	—	1	5	7	—
Richards	56	11	12	3	25	5
Adair and Dugan	28	13	3	3	5	4
Warren, Linton & Seannell	232	59	30	30	80	33
Total	2165	366(17%)	224(10%)	316(16%)	923(42%)	366(15%)

has sudden occlusion of the arterial blood supply to an organ—and particularly if he has atrial fibrillation or if an electrocardiogram indicates recent myocardial infarction

The majority of *peripheral* emboli tend to become arrested at certain locations (Fig. 55). The bifurcation of the femoral artery is in all large series the most common site of arrest of arterial emboli. This incidence ranges from approximately 25 to 40 per cent of all emboli reported.^{84 109 176}

This clinical fact is of great importance in the management of embolism since the femoral artery is readily approached under local anesthesia with minimal trauma to the already seriously ill patient. Next in order of frequency are emboli to the upper extremity the internal carotid artery and its branches, the popliteal artery the aortic bifurcation and the iliac bifurcation.^{84 109 177} Collected data are shown in Table 4.

Multiple and Repeated Emboli^{18 26 87 42 88 101, 107 148, 181 182} As is well known the patient with a mural ventricular thrombus or one in the fibrillating atrium may undergo successful peripheral embolectomy at one site, only to die abruptly of a second or third embolus to a vital center. Nevertheless numerous reports in the literature attest the fortunate fact that patients may survive multiple emboli at a single location even at the aortic bifurcation.^{168 178} and they may also survive removal of emboli from different vessels at the same time or at different times. All four extremities may be simultaneously or concurrently involved by emboli.¹ This type of phenomenon is of course not surprising in view of the nature of the thrombus in the heart from which particles

If the embolus is not removed within a reasonable period of time usually a relatively few hours thrombosis will occur both proximal and distal to the site of arrest of the embolus in many instances. If this proximal and distal thrombosis is sufficiently extensive successful operative intervention may not be possible. Fourth eventually the intima of the vessel involved by the embolus and the surrounding thrombosis may become seriously impaired and even should the embolus be removed these pathologic changes in the intima may render subsequent thrombosis at this site highly likely and frequently negate the results of embolectomy. However it has been pointed out by Shaw¹⁸⁴ that frequently the surrounding small vessels

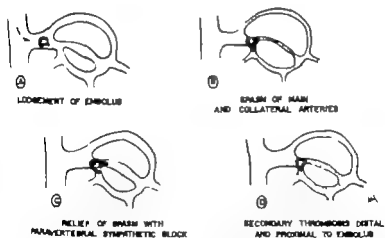


FIG 56 In addition to occlusion of the main arterial channel by the embolus, associated arteriospasm and later secondary thrombosis further reduce the blood supply to the part. Sympathetic nerve block may relieve arteriospasm but only prompt embolectomy can restore flow through the main channel and prevent or minimize thrombosis. (After Haimovici reference 56)

are not thrombosed in the presence of an embolus, and this factor is one which offers an opportunity to remove emboli and associated thrombosis by the retrograde flush technic even days following the initial embolism.

The mechanism by which the often severe vasospasm is produced is not entirely clear. While it may be largely a reflex type of vasospasm mediated via the sympathetic nerve fibers, humoral substances such as serotonin that may be liberated secondary to embolism must be considered as possibly playing a role. Since about three-fourths of all clinically evident arterial emboli lodge in limb arteries, sympathetic nerve block has been extensively used to treat the vasospasm¹²⁸ secondary to arterial embolism. In studying the collateral resistance in limbs with arterial obstruction, Dornhorst and Sharpey-Schaffer¹²⁹ found a spontaneous decrease in collateral resistance within a minute following acute occlusion of a normal artery, and in one subject there was evidence that the resistance continued to fall for a few days. It was found that sympathectomy was capable of causing a considerable decrease in collateral resistance both in acute and chronic occlusion, but in seven out of ten cases investigated this decrease was transient.

If severe ischemia of a part is not relieved, gangrene will occur. In deciding whether or not to intervene surgically, one should realize that if gangrene of, say, a leg is permitted to occur, the resulting operation for amputation of the extremity will be more traumatic to the patient than will prompt exploration of the femoral artery under local anesthesia.

DIAGNOSIS OF ARTERIAL EMBOLISM

The diagnosis of arterial embolism^{1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 51 52 53 54 55 56 57 58 59 60 61 62 63 64 65 66 67 68 69 70 71 72 73 74 75 76 77 78 79 80 81 82 83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 100} is usually made on the basis of the history and physical examination. Certain additional measures are of assistance of which angiography is perhaps the most important.^{1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 51 52 53 54 55 56 57 58 59 60 61 62 63 64 65 66 67 68 69 70 71 72 73 74 75 76 77 78 79 80 81 82 83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 100}

Characteristically the arrest of an embolus in a major artery results in marked symptoms of which pain may be the most striking. Yet this does not occur in all cases. In some patients there is a sudden onset of numbness and coldness where an extremity is involved but no pain. In still others the symptoms are of a progressive nature and only gradually does serious pain with numbness and coldness develop. A few patients apparently have a silent embolus in which no symptoms occur and here the evidence of peripheral arterial occlusion may be discovered only incidentally. In his series of 350 peripheral emboli Haimovici⁶⁶ was able to identify the mode of onset in 240 cases. A sudden onset was noted in 81.2 per cent, of which 59.5 per cent had sudden pain and 21.7 per cent had sudden numbness and coldness without initial pain. A progressive onset was noted in 11.7 per cent of which 7.5 per cent had gradual development of coldness and numbness. And in 5.7 per cent the arterial occlusion was apparently silent. He concluded that on the basis of these data arterial embolism would be suspected in 81.2 per cent of the cases or in about four out of five patients.

To recapitulate following sudden occlusion of a major artery to a limb the patient usually first experiences pain followed later by coldness numbness and pallor of the extremity collapse of the super

ficial veins, eventual loss of motor function, and finally ischemic changes which may progress to frank gangrene.

Physical examination reveals those objective findings just mentioned, as well as absent arterial pulsations distal to the level of the block. The embolus is most often located at the bifurcation of a vessel. The pulsations in all accessible arteries should be noted, since emboli may involve multiple sites. However, various peripheral pulses are at times absent in otherwise normal persons^{17, 116, 157}. The possibility of embolization should suggest cardiac disease, and atrial fibrillation should be excluded. Myocardial infarction may have produced physical evidence of heart failure, or fever, petechiae, an enlarged spleen and hematuria may indicate subacute bacterial endocarditis. An electrocardiogram may be helpful.

While as a rule the level of the arrest of the embolus can usually be rather well outlined by appropriate palpation for pulses, at times arterial spasm proximal to the site of arrest of the embolus may be so severe that one gains the impression that the occlusion is at a much higher level than it actually is. This error can also be made in interpreting arteriograms, since spasm or stagnant blood above the level of occlusion may result in the appearance of occlusion at a more proximal level⁶¹. In the occasional patient there may be difficulty in palpating the pulses in the leg because of edema, and in these patients oscillometry and angiography may be helpful. It is to be recalled that the abdominal aorta, iliac arteries and the supraclavicular portions of the

subclavian arteries can be palpated in patients who are not excessively obese.

The site of lodgment of the embolus is usually higher than the level of the change in temperature due to the collateral circulation. The change in color occurs distal to the change in temperature and often coincides with the portion of the extremity which will become gangrenous if the ischemia is not ameliorated (Fig. 57).

Clinical Pictures Further Comment.¹⁰⁸ The more common clinical forms met in peripheral arterial embolism are (1) embolism with marked ischemia and early death usually due to occlusion at the aortic bifurcation; (2) embolism followed by gangrene. This

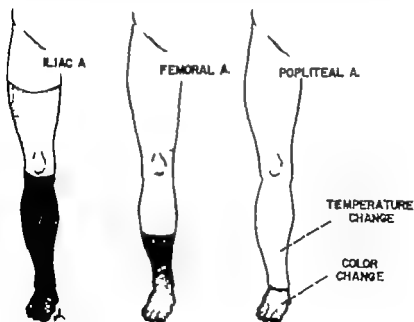


FIG. 57 The levels at which temperature and color changes appear respectively are dependent largely upon the level of arrest of the embolus.

is likely to be of the dry variety and may be observed in a few days following the onset of the embolic episode (3) Embolism with marked ischemia which subsequently subsides, resulting in chronic postembolic ischemia manifested by intermittent claudication, (4) marked embolism symptoms which subsequently subside, with no symptoms on exercise, and (5) silent or asymptomatic embolism which is discovered by chance, since the patient has been asymptomatic throughout ⁸⁵

Differential Diagnosis

The clinical picture of acute arterial embolism can be closely mimicked by several other conditions ⁸⁶

Venous Thrombosis ¹⁷¹ Simulating Arterial Embolization. Acute venous thrombosis can resemble arterial embolism to a remarkable degree. Waller ¹⁷² reported a case of venous thrombosis simulating arterial embolism with major gangrene. After amputation the extremity was carefully studied, and it was found that the gangrene of the leg had apparently been due to severe and continued arterial spasm in the presence of unsuspected thrombosis involving the femoral vein. The arteries through the extremity were patent. Similar reports have been made by Pringle, ¹⁷³ Humovici, ¹⁷⁴ Edwards ²² and Ebel and his associates ¹⁷⁵. Fountain and Taverner ¹⁷⁶ reported gangrene in three limbs resulting from venous occlusion.

The severe arterial spasm caused by the venous thrombosis may result in marked ischemia of the extremity prior to the onset of swelling manifested by edema which is so characteristic of venous occlusion. When this severe ischemia does precede the swelling

there is confusion regarding the diagnosis. After the edema due to venous occlusion has had time to occur however, there is much less likelihood that the venous occlusion will be mistaken for arterial occlusion.

Conversely acute arterial occlusion may simulate venous occlusion. Benton¹ reported a case in which femoral embolism closely simulated thrombophlebitis and the diagnosis of occlusion of the femoral artery was made only after several days and then by arteriography. In his case diffuse cyanosis and coldness of the entire left lower extremity were associated with moderate swelling and tenderness of the lateral aspect of the left thigh. The left thigh was 3.5 inches larger in circumference than was the right. Although the initial diagnosis was considered to be thrombophlebitis with arterial spasm, embolism to the femoral artery and arteriospasm secondary to trauma were considered as alternate possibilities. The present author was associated with Benton in taking care of this patient, and one can attest that there was difficulty in determining whether the primary fault lay with venous or with arterial thrombosis or occlusion. A subsequent venogram demonstrated patency of the venous system.

Arterial Embolism vs Arterial Thrombosis Another problem in differential diagnosis is that of distinguishing an arterial embolus from arterial thrombosis which has developed on the basis of an atherosclerotic plaque.¹²⁰ We have found it very hard in some patients to determine whether or not the acute vascular occlusion was due to arterial embolism or whether it was due to sudden complete thrombotic occlusion of an artery which had been partially occluded by athero-

sclerotic thrombus over a period of months or years. In fact, we have explored patients for suspected embolism only to find a very weak pulsation in the vessel and other evidence of chronic atherosclerotic occlusive disease of the terminal aorta and the common iliac arteries. Unfortunately, when the patient has had a recent coronary occlusion it is not always possible to determine whether or not embolism has occurred or whether, by fortuitous circumstance, complete thrombosis of a previously partially obstructed artery has occurred. Nevertheless, it has been pointed out previously that most patients with emboli will have evidence of heart disease. If such evidence is absent and if the patient is elderly, there is a good probability that sudden thrombotic occlusion has occurred. Warren and his associates¹⁷⁷ reported that the origin of emboli was clinically evident in 86.5 per cent of their patients, and this fact was an important aid in differentiating thrombosis from embolism. Aneurysms may become occluded by thrombus (Fig. 5b).

Acute arterial thrombosis may develop following trauma to the vessels, or due to infection. If it is apparent that gangrene of the extremity is threatened, it is better to explore for an embolus and be in error than to omit operation and have to amputate later where such could have been avoided.

Ischemic vs. Neurologic Pain. Patients with arterial disease producing ischemia are often first referred to a neurologist or a neurosurgeon. One patient of ours had been treated for "neuritis" prior to complete relief of his pain by aortic surgery. Gillfillan and his associates² reported a group of arterial occlusions that simulated neurologic disorders of the lower ex-



FIG. 58 This arteriogram reveals complete occlusion of a popliteal aneurysm by thrombus. Such thrombus can give off emboli to more distal vessels of the arterial bed

tr extremities Of course, ischemia of the extremities can unquestionably result in nerve damage, and this may be reflected in muscle atrophy—though the ischemia of the muscles themselves can and usually does produce such atrophy In one patient with late iliac embolic occlusion whom we treated, the first symptoms had appeared while he was sitting in a chair with his legs crossed following what had been diagnosed as an attack of “pancreatitis” The first manifestations were numbness and inability to move the extremity. Pain occurred late and was never severe He subsequently proved to have sustained a massive myocardial infarction, which explained the source of the embolus which originally had been thought to represent acute thrombosis The physician attending him at the time of onset of the embolism had felt that he was dealing with a major neurologic deficit and, despite the coldness and pallor of the extremity, the possibility of major arterial occlusive disease was not seriously considered until it was apparent that gangrene was far advanced

Arteriography⁵⁸ can be quite helpful in localizing the level of occlusion, and O’Connell and his associates¹⁻⁷ have used the segmental plethysmograph However, in our experience careful physical examination, with palpation for pulsations above and below the suspected level of arrest of the embolus, has been adequate to provide us with sufficient information to permit intelligent and effective operative intervention where this appeared to be indicated

MANAGEMENT OF ARTERIAL EMBOLI

The three basic problems presented by most patients with acute embolism are underlying heart dis-

case the ischemia produced by the embolus and the hazard of subsequent emboli.⁴ The management of arterial embolism consists of conservative measures, embolectomy, and follow up treatment with a view to preventing subsequent emboli.^{79 80 81 82 83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 100}

Conservative Measures

Conservative management^{2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 51 52 53 54 55 56 57 58 59 60 61 62 63 64 65 66 67 68 69 70 71 72 73 74 75 76 77 78 79 80 81 82 83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 100} includes appropriate measures aimed at treating the underlying cardiac condition which has produced the embolus as well as the changes produced by the embolus itself. The decision as to whether conservative or operative management is to be employed depends upon the basic underlying disease, the presence of other related or unrelated disease, the probable location of the arterial block, the degree of ischemia which has been produced, the time interval since occurrence of the embolic episode, and the facilities available.¹¹⁰ If the patient is in a hospital where arterial surgery is not performed and is in critical condition, every effort must of course be made to gain a successful issue with conservative measures.

Once heart disease has been evaluated and treated, further considerations in the general management of the patient with acute arterial block consist of efforts to improve the blood supply to the part. In the extremity this will include paravertebral sympathetic block to minimize arterial spasm. Papaverine has long been used to help relieve such spasm, but it appears to be used less frequently now than previously. Priscoline may be helpful. Heparinization should be instituted to reduce the degree of secondary thrombosis which may preclude the possibility of successful embolectomy at a later date, provided that the pa-

tient is too ill to be operated upon at once. Obviously one will not use anticoagulants if embolectomy is planned in the immediate future. The extremity is left at room temperature and the application of heating pads is scrupulously avoided. Smoking should be forbidden because of the vasospasm produced. The use of an oscillating bed or intermittent pressure changes⁶⁰ to provide passive assistance to blood flow has long been considered to be of value, but we have not used it for a number of years. It is our impression that the use of such beds has diminished considerably in the past decade. If paravertebral sympathetic block^{52, 60} proves efficacious, continuous spinal or nerve block anesthesia may be used^{10, 11, 12}. This method is in many respects superior to repeated paravertebral blocks, since one cannot be sure of always achieving an effective block. Similar anesthesia for the upper extremity has been described.⁶ Needless to say, there is risk of hemorrhage when blocks are performed in conjunction with anticoagulant therapy.¹²⁴ More recently fibrinolysis has been employed in the treatment of occlusive disease.¹⁶⁰

Of these nonsurgical measures, paravertebral sympathetic block or continuous spinal anesthesia, and anticoagulant therapy under proper circumstances, have proved in our experience to be the two most helpful measures available for diminishing the ischemia following acute embolic occlusion of major arteries in the extremities.

commonly used procedure.^{4 15 24 32 37 39 41 72 73, 74 87 92, 93 100 122 137 138 146 163} It has been emphasized previously that many factors enter into the decision of whether to perform embolectomy or to depend upon conservative measures in the given case. Moreover a considerable experience with both the natural history and prognosis of such patients in addition to technical proficiency in arterial surgery is often required for an optimal result. Obviously if satisfactory surgical facilities are not available conservative management will have to be employed regardless of other desiderata. Nevertheless there are certain features which are important in selecting the type of therapy best suited to the given patient. First the size of the artery involved will have an important bearing upon the selection of therapy. Many patients with a saddle embolus obstructing the bifurcation of the abdominal aorta will die if embolectomy cannot be successfully performed, but occlusion of the radial or ulnar artery or of the anterior or posterior tibial artery will not result in major gangrene or even in prolonged disability in most patients. Furthermore the incidence of successful removal of emboli from small vessels is not nearly as good with large vessels, other considerations aside.

Second when an embolus can be removed under only local anesthesia even in a critically ill patient, this should virtually always be done to reduce the stress produced by ischemia. In contrast, if spinal or general anesthesia must be employed one may on occasion consider exploration unjustified since the patient may have a slightly better chance of surviving if operation is not done. Fortunately the majority of

emboli involve the extremities, and femoral embolectomy under local anesthesia constitutes a very satisfactory procedure. This is far preferable to the shock of amputating a gangrenous extremity several days later.

Thus mature judgment is frequently called for in determining what course of action is most likely to achieve, first, a living patient and, second, a viable and useful extremity. It has been our impression that some surgeons are far too conservative in allowing precious hours to pass before an embolectomy is attempted. The golden time for successful removal of an embolus is within the first few hours following its occurrence. After 12 or more hours the formation of secondary thrombus and perhaps injury to the vessel wall may well preclude the restoration of normal pulsatile flow through the vessel involved. Therefore it is urged that the patient's total condition be evaluated promptly and a decision be made regarding whether or not it appears to be feasible to perform an embolectomy in the given case. If embolectomy is decided upon, it should be performed at the earliest feasible moment if the greatest chance of success is to be available to the surgeon.

Even where embolectomy might once have been feasible, it is of course contraindicated in patients who are virtually moribund, and in those where gangrene of the extremity is already far advanced.

Technic of Embolectomy

The details of embolectomy will of course vary according to the site at which the embolus has become arrested, but the general approach to the procedure is similar under most circumstances. In brief,

one uses suitable anesthesia local where possible to expose the artery. The technic of femoral embolotomy the most common site at which major emboli become arrested is shown in Figure 59. Control of the artery is gained both above and below the site of occlusion using tapes or catheters or suitable non-crushing clamps. The thrombus can usually be palpated through the vessel wall there is pulsation in the artery above the point of occlusion and no pulsation and a considerable diminution in the size of the artery below the point of occlusion. The vessel below the embolus is occluded and a longitudinal incision is made directly over the most distal portion of the embolus. Where one has a choice he should place the incision in the main artery proximal to its bifurcation since closure of the larger parent vessel is less likely to be followed by thrombosis than would closure of one of the two branches. When the incision has been made the force of blood pressure from above will usually cause the embolus to be flushed rapidly out of the artery. If it does not emerge immediately upon incision of the vessel it can be gently dislodged with a suitable instrument. It is important to be certain that no additional embolic material or thrombus remains in the vessel following removal of the main embolus. Furthermore as noted already the artery should be occluded below the level of the embolus to prevent the distal displacement of portions of the main embolus as it is being removed. The incision in the artery is then readily closed using a continuous over and over or baseball type of stitch of 4-0 or 5-0 arterial silk. If considerable arterial spasm is present one may inject 1 per cent procaine

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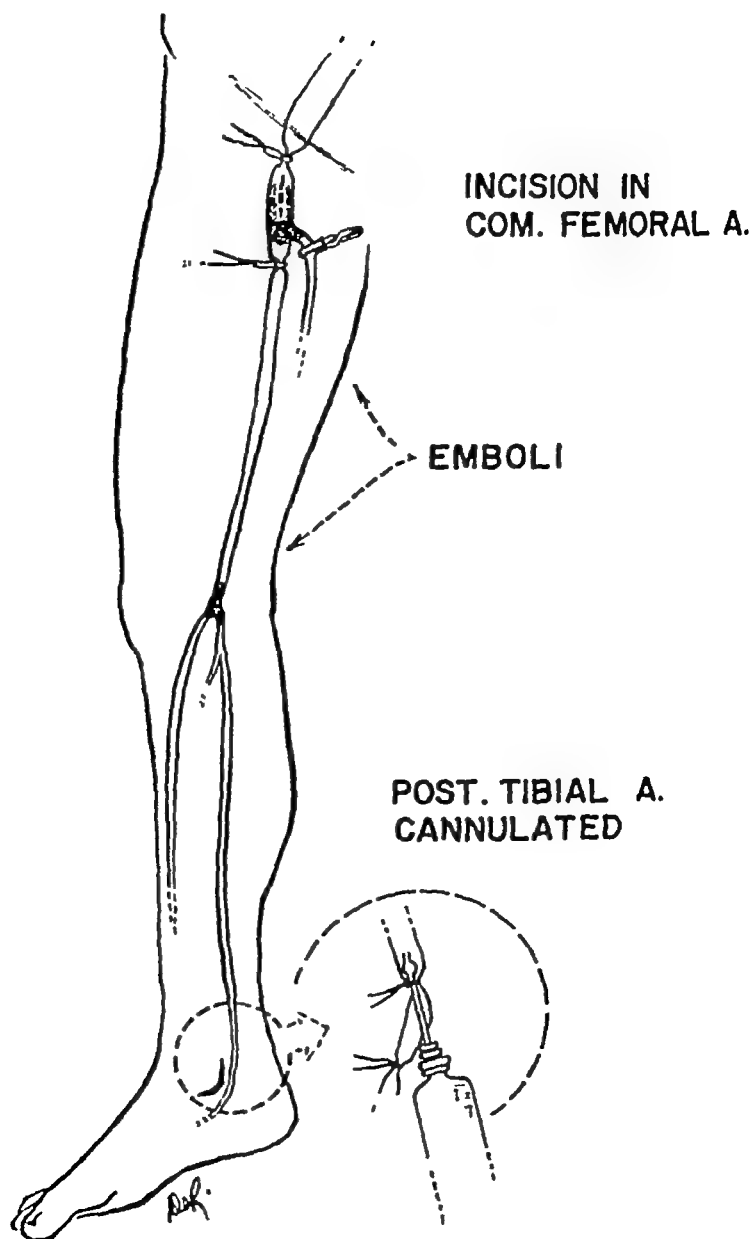


FIG. 59 Most arterial emboli become arrested at a bifurcation (see text). The common femoral artery, the vessel most frequently involved, is readily exposed under local anesthesia. After an incision has been made in the com

into the adventitia of the artery for a distance of 3 centimeters both above and below the level of arrest of the embolus. In our experience however satisfactory removal of the embolus with careful closure of the artery has been followed by good distal flow and continued pulsation where a subsequent embolization did not occur. We do often heparinize the patient when the wound can be effectively drained but with the passing of each year we are more and more convinced that careful arterial surgery is rewarded by good distal flow without the assistance of either anticoagulants or paravertebral sympathetic blocks in the vast majority of instances. Needless to say extremely meticulous hemostasis is essential if anticoagulant therapy is planned.

Late Embolectomy ^{11 22 123 133} The increased experience with arterial surgery that has accumulated in recent years has made clear the fact that frequently one can successfully perform embolectomy far later than had previously been thought. That is successful embolectomy with restoration of essentially normal arterial flow has been performed even several days following the original embolic episode. This is in sharp contrast to the widely honored surgical dictum to the effect that embolectomy must be performed within 12 hours following the onset of acute arterial occlusion. In analyzing the clinical and pathologic data which appeared to bear upon the possibility of successful late embolectomy Haimovici ²² concluded

mon femoral artery the force of the blood pressure from above will usually expel the clot. The proximal tape need not be tightened until the embolus has been removed, in most instances. The retrograde flush technic is also shown

that the following are especially important (1) persistence of an intima that is relatively damage-free, (2) the nonadherence of embolus and secondary thrombus to the intima, (3) a patent distal arterial tree prior to embolization and (4) pretreatment with anticoagulants. Thus it is to be emphasized that certain patients with persistent and severe postembolic ischemic manifestations may still be aided by late embolectomy regardless of the time which may have lapsed since the onset of the embolic episode, within limits.

The Retrograde Flush Technic The retrograde flush procedure, apparently first described by Leiman, Miller and Lund,⁹¹ has recently been revived and used successfully by a number of workers.²² This consists of exposing the artery far distal to the point of arrest of an embolus, and by means of a catheter or cannula flushing saline solution retrograde to cause the embolus and associated thrombus to be forced upward into the more proximal and thus larger portions of the artery, from which the clot is removed at the point of arteriotomy (Fig. 59). By using this maneuver it has frequently been possible to remove long segments of thrombus which would otherwise have precluded successful embolectomy. This has considerably extended the range of successful operative intervention.

To be sure, simple retrograde milking or massage of the artery⁷¹ may assist in displacement of distal embolus or thrombus backward to the site of arteriotomy. Furthermore, some workers have successfully expelled or displaced thrombi in a proximal direction by wrapping the leg with an Esmarch tourniquet from below upward. Additional maneuvers include

the use of catheter suction within the lumen of the vessel to aspirate clotted material. In general, we try to avoid excessive manipulation within the artery for damage to the intima constitutes an important cause of postoperative thrombosis.

PROGNOSIS IN ARTERIAL EMBOLISM

Even under the most favorable circumstances the results of either conservative (Table 5) or operative management of arterial embolism are frequently less satisfactory than might be expected. There are numerous reasons for this but the principal ones are the basic underlying heart disease which the patient has, the level of the arrest of the embolus and the circumstances under which the patient was treated. With respect to the last, a major factor in salvage of the extremity is the length of time which has elapsed prior to embolectomy when such therapy is indicated and is employed. It has been emphasized that there may be multiple simultaneous emboli to different sites in the body; there may be embolization to inaccessible vessels such as the more distal portions of an internal carotid or a coronary artery and subsequent embolization may occur days, weeks or months following the initial one. When general anesthesia must be employed for the removal of an embolus additional stress is placed upon a critically ill patient in many instances and this may prove fatal. Too the removal of the embolus from the artery may be followed by thrombosis at the arteriotomy site with subsequent gangrene. Heparin and dicumarol-like drugs can be used to prevent postoperative thrombosis, but this is not always effective. Furthermore they impose the hazards of

TABLE 5

Clinical Course of Untreated and Conservatively Treated Peripheral Arterial Emboli in 300 Cases

Site of Occlusion	Embolism with Marked Ischemia and Early Death		Embolism followed by (gangrene)	Chronic Post-embolic Ischemia	Anischemic Embolism	Embolism with Undetermined Degree of Recovery		Totals
	with Marked Ischemia and Early Death	Embolism followed by (gangrene)				Embolism	Silent Embolism	
Aorta, bif	8	11	7	7	—	2	—	28
Iliac, com	3	25	7	1	5	3	—	43
Iliac, ext	1	31	21	17	17	20	—	10
Femoral	17	3	16	4	4	—	—	108
Popliteal	2	1	—	—	4	1	—	47
Distal post.	—	—	1	1	6	2	—	9
Distal ant	—	—	1	1	17	—	—	9
Artery	4	1	—	1	4	—	—	13
Chilid	4	—	—	—	17	—	—	25
Idiopathic	—	—	—	—	4	—	—	4
Unlabeled	—	—	—	—	1	—	2	4
Total	39	81	55	76	32	17	300	100

Percentage of cases with embolism 128 (42.7%)

Percentage of cases with anischemic embolism 128 (42.7%)

Percentage of cases with chronic post-embolic ischemia 128 (42.7%)

spontaneous hemorrhage. In general the older the patient the less satisfactory are the results of embolectomy.¹⁴⁴ Patients who have embolism secondary to rheumatic heart disease with atrial fibrillation have a better prognosis than those whose embolism is due to myocardial infarction or atherosclerotic heart disease with atrial fibrillation and unquestionably the experience of the surgeon performing embolectomy is an important factor in a successful outcome. Of course in some instances it is not possible successfully to remove the embolus regardless of the technic or approach employed.

Limb Survival.¹⁷⁶ In reviewing their cases Warren Linton and Scannell¹⁷⁷ found that there was an 85.7 per cent limb survival rate for surgical as opposed to a 60.5 per cent rate for nonsurgical treatment in a series of patients studied from 1937 to 1946. In a second series studied from 1946 through 1953 these figures were 83.3 per cent and 88.5 per cent respectively. Nevertheless, these workers pointed out that one cannot conclude that nonoperative is more effective than operative treatment for surgical removal of the embolus was undertaken in 47.7 per cent of the cases in the 1946-1953 series (Series II) as opposed to only 22.7 per cent of the cases in series I which extended from 1937 to 1946. Rather the inference was that an earlier referral of the patient to the hospital with the resulting wide option on the part of the doctors to select the proper type of treatment had been prevalent in the later period of study. Thus an extension of surgical management to a larger series of cases had resulted in relegating patients not operated upon to those with occlusions of relatively minor ar

teries. The most important conclusion to be drawn from this group was that, by early referral, the number of patients in whom it was possible to perform surgery prior to irreversible changes in the extremity was substantially increased.

Mortality. The over-all mortality rate of their patients with arterial embolism to the limbs treated during the period of 1937 to 1953 was 30.6 per cent, as reported by Warren, Linton and Scannell¹⁷⁷. This was an average of the 35.3 per cent mortality in the groups studied from 1937 to 1946, as compared with a 27.8 per cent mortality rate in the group studied from 1946 to 1953. These workers pointed out that (1) cerebral and mesenteric embolism occurring concomitantly with that to the limbs, (2) progressive impairment of cardiac function due to myocardial infarction, and (3) cardiac decompensation due to valvular disease render patients with these complications particularly vulnerable to subsequent and often fatal episodes (Table 6). The reduction in the mortality rate from

TABLE 6 *Causes of 67 Deaths* in 200 Patients with Arterial Embolism*

Cerebral Embolism	20	30.0%
Cardiac Failure	19	28.3%
Mesenteric Embolism	11	16.4%
Myocardial Infarction	7	10.5%
Cerebral and Mesenteric Embolism	1	1.4%
Pulmonary Embolism	2	2.9%
Other**	7	10.5%

* Only deaths occurring on the same hospital admission as an embolism are included.

** Bacterial endocarditis¹, gastro intestinal hemorrhage¹, aspiration vomitus¹, ulcerative colitis¹, unknown².

(From Warren, R., Linton, R. R., and Scannell, J. C. Arterial embolism, recent progress. *Ann Surg.* 140: 311, 1951.)

the first group to the second was attributed in part to a slightly lower incidence of cerebral and mesenteric embolism to a better condition of the patients on arrival at the hospital and to a decrease in the complications resulting from massive ischemia of the lower limbs.

Limb Survival According to Embolic Site. In aortic, iliac, and femoral embolism embolectomy is superior to conservative treatment. In other words the incidence of limb gangrene and the patient mortality rate increase in direct proportion to the size of the vessel occluded. Thus the total salvage rate in aortic and iliac occlusion is less than in femoral and popliteal occlusion. Warren and his associates¹⁷⁷ found that occlusion of the popliteal artery by embolism carried a better immediate prognosis than occlusion of the artery due to thrombosis or trauma since occlusion by an embolus usually occurred distal to the origin of collateral branches.

Perhaps the most important conclusion to be drawn from these data is that regardless of how optimal the therapy is the mortality rate among patients with major arterial embolism is still disturbingly high and the late salvage leaves much to be desired. In Haimovici's⁴⁶ survey of 330 unselected cases of embolism of the extremities a total of 61 out of 128 untreated and conservatively treated cases died in the hospital a mortality of 47.6 per cent. The surgically treated cases showed a mortality of 51.7 per cent, a rate only slightly above the medically managed cases. Nevertheless it should be realized that surgical therapy doubtless was used in patients who were otherwise not expected to survive major arterial occlusion. Such

figures merely emphasize the importance of individualization of cases with respect to therapy

In discussing the results of direct arterial surgery as compared with medical management of patients with arterial embolism, Shumacker and Jacobson¹⁶⁵ expressed the opinion that there was little merit in trying to compare such groups. Those operated upon were considered generally to include only patients with eminent threat of gangrene, as was true of all but three of their cases, or marked ischemia with rest pain and anesthesia. In the group not operated upon there was a high proportion of patients who were either admitted to the hospital late with well-established gangrene or who had promptly undergone spontaneous improvement in circulation to such a degree as to make it evident that the limb had an excellent chance for survival under any circumstances. A review of their cases resulted in the conclusion that the over-all results were much better in the patients who were operated upon than in those who were not, where similar degrees of ischemia existed. It was believed that the mortality in patients with embolism to the aorta and peripheral vessels, excluding only those with embolism to arteries supplying the brain and viscera, was related to the age of the patient and to the underlying disease. *The mortality rate was approximately twice as great in patients over the age of 60 as in those younger.* The mortality was relatively small in patients with rheumatic heart disease but considerably higher in patients with myocardial infarction, arteriosclerotic heart disease, and hypertensive cardiovascular disease. It was again emphasized that the results of embolectomy are better the earlier

the embolectomy is carried out and that following embolism the survival of the limb is primarily dependent upon the presence or absence of propagating thrombus in the arterial tree. Shumacker and Jacobson¹⁴⁴ as well as others¹⁷⁷ have emphasized the fallacy of the sense of marked optimism which appears to exist among many physicians concerning the prognosis in cases of embolism to the main arteries of the upper extremity. In the group not treated by embolectomy there were four cases of brachial one case of axillary and one case of subclavian embolism. Amputation was required in four of the six patients a fifth patient died but with apparently satisfactory circulation. Shumacker and Jacobson¹⁴⁴ achieved an overall survival rate of limbs of 79 per cent as compared with 83.3 per cent reported by Warren and his associates¹⁷⁷ and 58.5 per cent reported by Dye and his colleagues.⁹⁸ Haimovici⁴⁴ reported a limb survival rate of 48.2 per cent but his cases were perhaps more comprehensive in scope and were unselected.

The late results of arterial embolectomy were assessed by Goldowsky and Bowen⁸² on the basis of 46 operations performed on 42 patients over a period of 11 years. The overall operative mortality for peripheral arterial embolectomy was 37 per cent. The chances of surviving the first embolectomy were 61.9 per cent. The chances of surviving the first embolectomy and living three years or longer were 44.4 per cent. The chances of surviving the first embolectomy and living five years or longer were 40 per cent. They emphasized that the initial high mortality rate still constitutes a serious problem though the limb

salvage rate has improved and is generally satisfactory. It was believed that long-term postoperative anticoagulant therapy, first with heparin and later with oral dicumarol, had been definitely beneficial.

Prophylaxis Against Subsequent Emboli

Inasmuch as the late results of embolectomy are frequently poor due to the fact that subsequent and fatal emboli occur, it is of the greatest importance to explore means by which subsequent emboli can be prevented. The use of quinidine to control atrial fibrillation⁷⁸ and other prophylactic measures have been somewhat disappointing.

Operative Measures For a time there was considerable interest in the excision of the *atrial appendix* in patients with atrial fibrillation who had thrown emboli to various arteries,^{11, 65, 102, 120} for patients with this condition commonly exhibit embolic phenomena before, during or following mitral commissurotomy.^{14, 23, 49, 51, 53, 66, 75, 113, 121, 168} However, it is our impression that excision of the atrial appendix in fibrillating patients who have thrown emboli is less commonly employed at the present time than it was several years ago. Acute bacterial endocarditis is of course treated with both massive antibiotic therapy and anticoagulants, with a view to both curing the infection and diminishing the number of emboli. Myocardial infarction with mural thrombosis, with or without aneurysm of the ventricle, is treated according to the given case. It is of interest to note that a number of ventricular aneurysms have now been successfully resected and the defect closed, with subsequent improvement in the cardiovascular function.

tion of the patient. Such ventricular aneurysms¹¹⁸ commonly contain considerable amounts of clot which may give rise to emboli at any time. Aneurysms of the aorta—whether fusiform, saccular or dissecting in type—may give rise to emboli. These may or may not be susceptible to surgical management. A cervical rib which is producing thrombosis with distal embolization of parts of the thrombus is managed according to the method best suited to the given case.

Long Term Anticoagulant Therapy^{108 117 119 159} Protracted anticoagulant therapy to prevent embolism has been employed for some years.^{8 48 166} and Wood and Conn¹⁰⁸ have reviewed its use in chronic rheumatic heart disease. Seven ambulatory clinic patients with mitral stenosis and chronic atrial fibrillation were described who had previously had systemic arterial embolism. They had been maintained on anticoagulant therapy for periods ranging from 20 to 48 months without a definitely established subsequent embolism. However one patient had had an episode of confusion and apparent slight right sided weakness which had cleared quickly but which the authors believed had probably been due to a small embolic episode.

Certainly it has been established that anticoagulant drugs can be administered to the ambulatory patient over long periods of time without prohibitive risk of hemorrhage. Such hemorrhage does occur however and we have treated a patient who developed a large retroperitoneal hematoma that was believed to represent a ruptured abdominal aneurysm but which proved to be a massive hematoma apparently secondary to a long acting heparin like compound which he

was receiving as treatment for a mild attack of hemiplegia sustained some weeks earlier. Thus the overall experience suggests that prolonged anticoagulant therapy does provide significant protection against recurrent embolism and within the limits of acceptable therapeutic risk.

EMBOLI TO SPECIFIC ARTERIES FURTHER COMMENT

Internal Carotid Artery. The internal carotid artery and its branches were the site of 50 (14.6%) of 337 emboli reported by Warren, Linton and Scannell¹⁴. Haimovici⁵⁰ reported an incidence of 31 per cent involvement of cerebral arteries in 147 visceral emboli (which excluded emboli to the extremities). Therefore it is clear that the cerebral vessels are involved in a very considerable percentage of arterial emboli. Should carotid arteriograms disclose obstruction at the carotid bifurcation, an effort should be made to remove the embolus, though attempts to remove cerebral emboli have not been conspicuously successful.

Emboli to Arteries of the Upper Extremity.¹⁴⁴ The management of emboli to the arteries of the upper extremity has been touched upon previously and we shall only repeat that such emboli are far from benign. Approximately 17.5 per cent of 337 emboli reviewed by Warren and his associates¹⁵⁷ involved the arteries of the upper extremity. Shumacker and his associates¹⁵⁵ and others have emphasized the fact that such emboli frequently result in gangrene and that they should not be looked upon as benign sites of arterial occlusion following which most limbs may

be expected to survive without serious residual ischemia. Furthermore emboli to the upper extremity are particularly accessible to removal under local anesthesia, especially those distal to the clavicle. Most of these arteries are readily exposed under local anesthesia using short longitudinal incisions directly over the vessel. Therefore they should be removed promptly to restore normal blood flow through the arm. We have treated a number of patients in recent years who had sustained occlusion of arteries of the arm with subsequent relative ischemia. They had either intermittent claudication on exercise of the arm or actual gangrene for which amputation was required. The value of cervical sympathectomy in patients in whom normal pulsatile flow cannot be restored through successful embolectomy should be borne in mind. It is true of course that the collateral blood supply of the upper extremity is extensive, and this limb is more likely to survive an embolus than are portions of the lower extremity following occlusion of the artery at important levels. The occlusion of the brachial artery below the origin of the profunda branch is less serious than occlusion of the brachial artery just above the offtake of this important collateral supply.²⁸

Visceral Arteries Within the Abdomen.⁸ Emboli to the celiac, superior mesenteric,^{9, 10} and renal arteries are not rare. Shaw and Rutledge¹⁸⁴ reported a superior mesenteric artery embolectomy in the treatment of massive mesenteric infarction. A prompt operation and exposure of the superior mesenteric arteries should be followed by successful embolectomy in many instances. Miller and DiMare¹¹² reported

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a successful case of superior-mesenteric-artery embolectomy and small-bowel resection with recovery. Therefore, the previous practice of simply resecting obviously ischemic bowel, with resulting small-bowel insufficiency, should be abandoned where possible, in favor of an earnest effort at re-establishing an adequate pulsatile blood flow through the superior mesenteric artery by embolectomy and removal of adjacent thrombus.

Kaiser and Ross⁹⁷ reported total renal infarction from bilateral arterial emboli. To be sure, the kidneys are very commonly affected by embolization, particularly in association with the vegetations of bacterial endocarditis. In fact, the finding of red cells in the urine, reflecting emboli to the kidneys with infarction, is one of the common manifestations of endocarditis. In addition, emboli from thrombus are frequently dislodged into the renal arteries in the course of thromboendarterectomy for lower aortic occlusive disease due to atheromatous material. We routinely palpate the renal arteries following completion of the proximal anastomosis after an aneurysm has been resected just below the renal arteries, to be certain that no thrombus has been displaced into the renal arteries. Embolization to the kidney can produce hypertension.¹⁰¹ Renal infarction frequently results in pain in the flank.

The Aortic Bifurcation and the Iliac Arteries.^{20, 21, 27, 77, 97, 114, 118, 131, 134, 145, 158, 161} Approximately 9 per cent of peripheral arterial emboli become arrested at the aortic bifurcation. Furthermore, this massive obstruction to blood flow represents a physiologic circumstance which will not be tolerated by most individuals.

and will often terminate in death unless it can be relieved surgically. The approaches by means of which aortic embolectomy may be accomplished are shown in Figures 59 and 60. One may elect either to approach the aorta through a retroperitoneal or a direct lower abdominal mid line incision or he may use a more

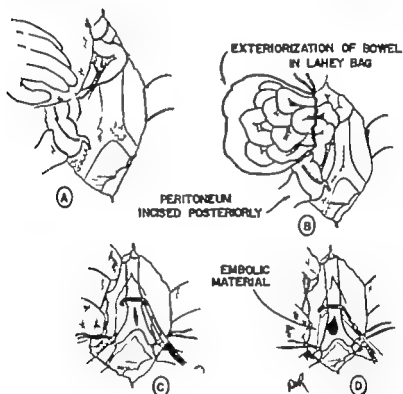


FIG 60 The lower aorta is perhaps best exposed through a mid line abdominal incision bearing in mind that the bifurcation of the aorta is at approximately the level of the umbilicus. The use of a Lahey bag for retraction of the small bowel is recommended. Either a Satinsky clamp as shown or a vertically applied Crafoord clamp is satisfactory for control of the aorta proximal to the embolus at the bifurcation.

conservative exposure and attempt to remove the embolus from the bifurcation of the aorta by passing catheters up each femoral artery, through which the embolus is aspirated. The latter was the preferred approach for many years, but was later superseded by the direct transabdominal approach. More recently, however, the bilateral femoral approach has been revived by Willman and Hanlon¹⁸². They regarded this

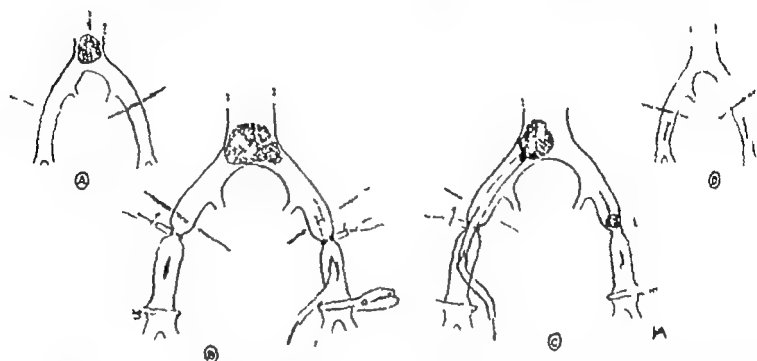


FIG 61 Saddle emboli of the aorta can at times be removed satisfactorily under local anesthesia using a retrograde femoral approach. Exposure and occlusion of each femoral artery is required to prevent dislodgement of embolic material into more peripheral arteries.

approach as a safer operation for aortic embolism and reported four consecutive successful embolectomies by way of the femoral arteries under local anesthesia. Since patients with recent myocardial infarction and mural thrombosis are in especially poor physical condition in many instances, the possibility of using only local anesthesia to approach the embolus through the femoral arteries from below is certainly appealing. Unfortunately, in our experience this procedure is not always satisfactory and one must then give the patient either spinal or general anesthesia and explore the

abdomen after all. This second exploration has usually followed a considerable period of time during which attempts were made to remove the saddle embolus from below. Yet conservatism in such patients has much to recommend it, and these authors have made a good case for re-examination of the femoral approach. In performing aortic or other embolectomy it is important to occlude the vessels below the site of arrest of the embolus to avoid distal embolization as the embolus is being removed. The use of a Lahey bag²² for small bowel retraction facilitates transabdominal exposure (Fig 60).

The Femoral Artery The femoral artery is the site most frequently involved by emboli. This is a fortunate circumstance since the femoral artery is readily approached under local anesthesia and femoral arterial embolectomy is truly a simple procedure. The technic is shown in Figure 59 and little further discussion is required. Suffice it to say that a saddle embolus at the bifurcation of the femoral artery is very likely to result in gangrene of a portion of the distal extremity unless successful embolectomy is performed. It is far preferable to remove the embolus under local anesthesia subjecting the patient to very little operative stress than it is to have to amputate the leg later in a patient whose condition has deteriorated still further.

The Popliteal Artery Considerable emphasis has been placed recently on vigorous attempts to relieve sudden occlusions of the popliteal artery by operative intervention. Roberts and Davis²³ reviewed this problem and reported seven cases of acute popliteal

and Knowles⁴⁸ have emphasized the anterior tibial syndrome due to such arterial embolism and thrombosis. Ischemic necrosis of the muscles of the anterior tibial compartment had been a notable finding in the group of patients which they reported.

Summary and Conclusions

1 Arterial embolism constitutes a serious clinical problem which often results in loss of limb or life.

2 Particles of thrombus account for the overwhelming majority of arterial emboli, but other substances such as air, fat, atherosclerotic plaques, tumor cell masses from the heart and metal projectiles may also enter the systemic arterial tree.

3 Most arterial emboli arise in the left side of the heart—atrium, mitral valve or ventricle—but they may also arise in association with various types of aneurysms, cervical rib compression, arterial trauma with thrombosis, aortic surgery, intra-cardiac surgery, and from venous clots which pass through atrial or ventricular septal defects.

4 Virtually any artery of the body may be the site of arrest of an embolus. The incidence at different sites has been presented, the femoral artery being the one most frequently involved. Emboli may be multiple or recurrent, and repeated embolization to the same site is not rare.

5 The ischemia produced by the lodgement of an embolus is due to occlusion of the main arterial channel, associated spasm of the main and collateral vessels and, later, secondary thrombosis proximal and distal to the embolus.

6. The diagnosis of arterial embolism rests largely

upon the history associated evidence such as that of heart disease which might explain the source of an embolus and the physical findings of ischemia of the involved part. Arteriography is helpful upon occasion but is usually unnecessary. The condition must be differentiated from acute arterial thrombosis, acute venous disease and nerve lesions.

7 The management of arterial embolism includes treatment of underlying heart disease, relief of ischemia and prevention of subsequent emboli. The considerable mortality associated with arterial embolism is due largely to heart failure, to the immediate stress of the current episode of ischemia and to subsequent emboli. Conservative measures are outlined. Embolectomy under local anesthesia is usually a satisfactory procedure when arteries to the limbs are involved. Even saddle emboli to the aorta or iliac arteries can be so removed in a retrograde fashion using a bilateral femoral approach. Techniques of embolectomy are described. The vast majority of emboli involve peripheral vessels though the coronary arteries and those to abdominal viscera are involved upon occasion.

9 The prognosis for limb survival in arterial embolism is good (approximately 75 per cent) under experienced management, provided that treatment can be instituted promptly. The acute episode with associated problems carries an overall mortality rate of approximately 35 per cent.

10 Long term prophylactic anticoagulant therapy is only partially effective in preventing subsequent emboli.

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CHAPTER 7

Traumatic Injuries

ARTERIAL injuries have challenged the best efforts of surgeons down through the ages. An important segment of the medical history of each war has been devoted to an analysis of the incidence and types of arterial injuries and to the amputation rates associated with the interruption of arteries to the extremities.^{1 2 10 20 32} But although the records of the successive major conflicts have reflected an increasing preoccupation with the study and improvement of methods designed to treat arterial trauma more effectively by restorative surgery^{36 39 106} it was not until the recent Korean War that immediate repair by direct suture was widely performed under field conditions. The success achieved with this prompt restitution of arterial continuity has revolutionized the management of arterial injuries not only in battle casualties but in civilian life as well.

In this final chapter the incidence, types, complications, diagnosis and treatment of arterial injuries will be presented as follows:

I. *Historical Considerations*

A. General Comment

II. Amputation Rates in Wars Prior to 1950

C. The Korean War Experience

II *Types of Arterial Injury*

- A. Contusion with Spasm and/or Thrombosis
- B. Simple Laceration with Incomplete or Complete Division
- C. Loss of Arterial Substance
- D. Aneurysms—True and False
- E. Arteriovenous Fistula
- F. Causalgia

III. *Diagnosis of Arterial Injuries*IV *Management of Arterial Injuries*

- A. General Considerations
- B. Specific Types of Injury

HISTORICAL CONSIDERATIONS

General Comment

Injuries to major arteries are frequent in warfare, but they are not uncommon in civilian hospitals to which trauma cases are referred. The incidence of major arterial wounds among battle casualties in the Crimean War (1854–1855), American Civil War (1861–1865), Russo-Japanese War (1901), World War I (1914–1918), and World War II (1941–1945) was on the order of 1 per cent.²⁰ Moreover, the frequency of injuries to particular arteries and the amputation rates resulting from injuries to various arteries were remarkably consistent throughout the various conflicts up to the American experience in the Korean War.

Amputation Rates in Wars Prior to 1950

Unlike the patient with a simple knife laceration of a vessel that may be encountered in civilian practice, the wounded soldier may have not only an ex-

tensively damaged artery but also major bone muscle and nerve damage which renders arterial reconstruction futile.²⁰ In discussing reasons why the most meticulous and careful arterial surgery could be successful in only a relatively small percentage of extremity injuries under conditions of warfare DelBakey and Simeone²¹ pointed out in an analysis of 2171 cases of arterial injuries in World War II that 68.6 per cent of the amputations had to be performed for massive trauma 19.5 per cent because of vascular injury *per se* and 11.9 per cent for infections such as clostridial myositis and other types of bacterial invasion. Massive soft tissue destruction in association with arterial trauma is not common in civilian life since the injury has usually been inflicted with either a relatively low velocity bullet or with a sharp object such as a knife. Moreover infection is much less frequently a serious problem in such civilian injuries.

The amputation rates recorded following trauma to individual major arteries prior to the Korean War are shown in Figure 6.2. In addition to the different rates for amputation which followed injury to specific arteries numerous other factors entered into the decision of whether or not amputation had to be performed in the given case. These factors included shock the magnitude of other injuries as discussed previously the delay involved in getting the patient to surgery²² associated infection and other problems. Nevertheless with these other variables in mind it is worth examining the incidence of amputation which follows ligation of a given major artery. Such information is of assistance in the selection of optimal treatment for an arterial injury where several

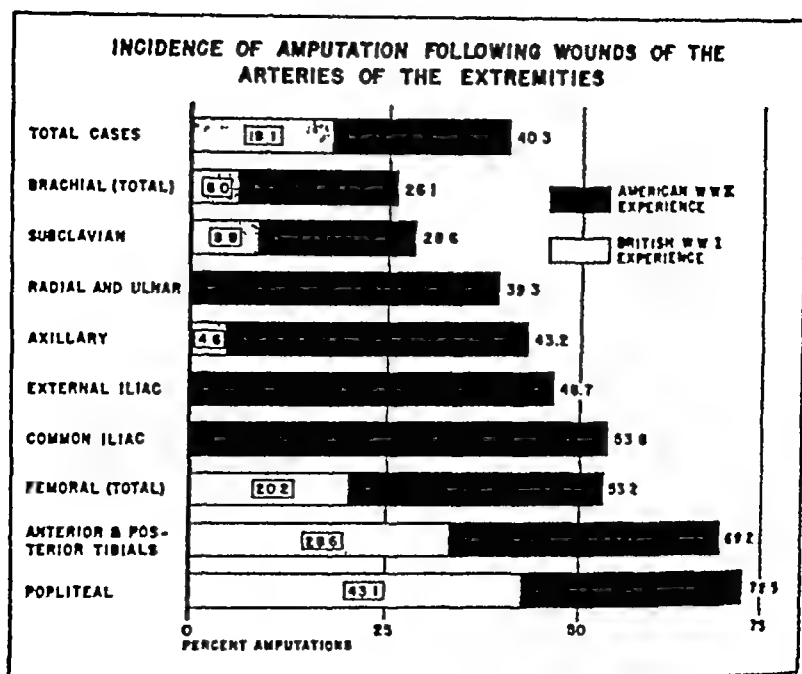


FIG. 62 There is a notable similarity in the rates recorded for the different conflicts (De Bakey, M. I. and Simeone, F. A., Ann Surg 123:531, 1946)

factors must be considered, one would accept somewhat more operative risk in repairing an artery whose occlusion would lead to gangrene of the extremity in a high percentage of cases than he would for the repair of an artery whose simple ligation would result in gangrene in a low percentage of cases. However, the modern concept of arterial management is to restore pulsatile flow through all but the very small arteries where this can be accomplished without prohibitive risk to life. Figure 62 shows that in the wars indicated, injury to the brachial artery was associated with an amputation rate of 26.1 per cent. In contrast, injury to the popliteal artery was associated with an

amputation rate of 72.5 per cent. Injury and occlusion of blood flow through other arteries of each extremity were associated with intermediate percentages for amputation rates. Incidentally it was in the primary repair of popliteal arterial injuries that the advances in the recent Korean War were so striking, the amputation rate for popliteal artery injuries being reduced to 32.4 per cent when the vessel was repaired as compared with 72.7 per cent for those ligated.²²

Relation of Treatment Time Lag to Amputation Rate As with other types of arterial disease particularly embolism the time which elapses between the onset of ischemia due to interruption of pulsatile arterial flow and the time of operative correction has a significant effect upon the amputation rate. For example, in the American experience in World War II it was found that the amputation rate for casualties reaching surgery within ten hours following injury was 36.7 per cent for those reaching surgery in the period from ten to 20 hours 48.8 per cent, and for those coming to surgery after 20 hours 63.0 per cent.²³ These figures were of course collected prior to the large scale and successful efforts at arterial repair that were developed in the Korean War. The higher amputation rate following injury to a given artery because of greater delay in bringing the patient to operation was due to prolonged shock which of course intensified the ischemia of the extremity whose main arterial blood supply had been interrupted to increased infection due to delay in debridement of the wound and to the general deterioration in the patient's condition due to prolonged exposure and inadequate treatment—factors previously mentioned.

It will be seen subsequently that the type of damage to an artery also has an important bearing upon the therapeutic results achieved. Simple lacerations that are readily repaired are associated with a lower incidence of gangrene in both military and civilian practice than are extensive arterial contusions which result in thrombosis of not only the main arterial trunk but of collateral branches as well. Whereas the simple laceration may be easily sutured, the thrombus that has propagated in both a proximal and distal direction from the site of injury may not be removed completely. Other types of arterial damage with resulting spasm and related phenomena occupy an intermediate position in the frequency with which they result in amputation.

The Korean War Experience

The statistics gathered regarding the results of management of arterial injuries in wars prior to the Korean War, including World War II, served as a valuable baseline against which to compare the substantial advances brought about by the application of direct arterial suture methods¹⁴ in the Korean War. The results achieved of course reflected the intense preoccupation with cardiovascular surgery in general which had developed shortly following World War II. The modern knowledge of arterial physiology, methods of suture, instrumentation developed specifically to permit gentle handling of arteries, and increased understanding of the technical requirements necessary to permit maintenance of arterial flow all were available to the surgeon. The results were to effect the application of these methods during

ing the Korean conflict. The demonstration on a relatively large scale that arterial repair could readily be performed under field conditions⁶⁷⁻¹¹¹ with excellent results led to prompt adoption of arterial reconstruction techniques in civilian hospitals throughout the United States with equally gratifying results. Within a decade it has become rare that simple ligation of a major arterial trunk is justified—for either transverse suture repair of an incomplete laceration or direct anastomosis of a divided vessel or the insertion of a prosthesis to bridge the gap left by the destruction of a considerable amount of the arterial tissue can now be done. This advance in the technical aspects of arterial surgery will be further enhanced by subsequent advances in the general supportive therapy of patients in the management of fractures and soft tissue healing and in the control of infection.

TYPES OF ARTERIAL INJURY

The various types of injury which may be inflicted upon major arteries will be discussed in some detail subsequently, but a few comments regarding the anatomic and functional defects that may be produced (Fig 68) are in order here. Most of the lesions to be considered will have been produced by blunt trauma, glass or knife or gunshot wounds. Although other insults such as cold injury and electrical burns are important, these forms of arterial trauma will not be reviewed here.

Contusion of an artery may be followed by severe arterial spasm^{12-14, 71-73} with or without thrombosis. Thrombosis is a particularly serious complication for it may result in occlusion not only of an extensive

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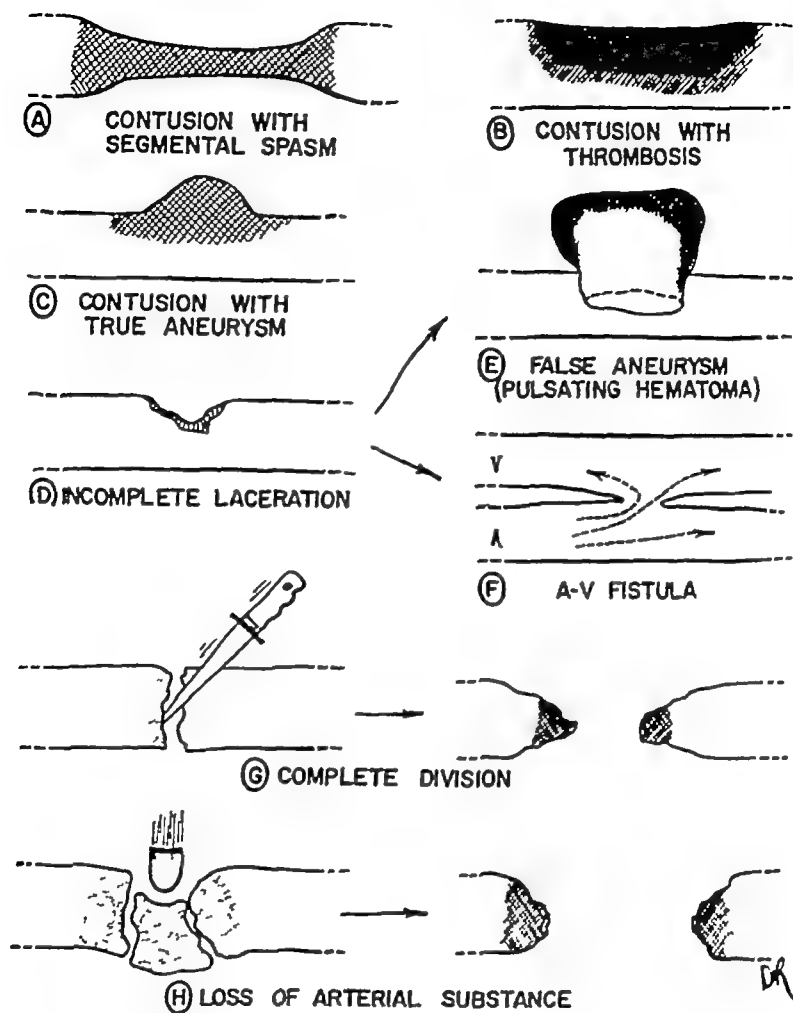


FIG 63 Comparison of major types

segment of the main trunk but also of numerous collateral branches as well. Loss of collateral blood flow is of course particularly likely to result in gangrene of the part when the main trunk has been blocked. Contusion may be produced in numerous ways, including gunshot wounds, blunt trauma⁹⁹ causing fractures and dislocations, and even chronic trauma

such as is sustained by the axillary artery of a person using crutches improperly.⁸¹

Laceration of a vessel may range from a simple incomplete tear to complete division of the artery. These injuries may be caused by gunshot wounds penetrating sharp objects or bone fragments in the presence of a fracture to mention a few. A major complication of arterial laceration is external blood loss plus the hematoma which forms within the tissues of the wound. Relatively great volumes of blood may be lost into a thigh for example not to mention the massive bleeding which may occur into one of the coelomic cavities. The aorta itself may be injured and although such patients usually do not survive to reach the hospital a number of instances of successful repair of aortic lacerations have been reported.⁸²⁻⁸⁴ Those patients who did survive the immediate injury have frequently formed pulsating hematomas for example in the thorax.¹⁰³

Loss of arterial substance confronts the surgeon with a more difficult problem than does a simple laceration. If only a short segment of arterial wall has been destroyed or must be debrided (Fig 63) it may be possible to bring the ends of the artery together for successful anastomosis. This is facilitated where necessary by mobilizing the vessel for a number of centimeters on either side of the injury. In one case we treated it was found possible to perform an anastomosis of the popliteal artery without excessive tension only after flexing the knee on the thigh. It was then maintained in this position with a posterior splint for seven days following surgery (Fig 64). Thereafter the cast was removed and the

leg was gradually extended until full motion was achieved, no decrease in the foot pulses occurred

In other instances, however, the extensive loss of arterial tissue renders grafting with homograft, saphenous vein, or synthetic material essential to permit restoration of arterial flow. Traumatic aneurysms may represent either true or false aneurysms (pulsating hematomas). In the first instance at least a portion of the arterial wall is still intact, whereas with a false aneurysm continuity of the arterial wall has been lost at the site of the aneurysm, here the so-called aneurysmal sac merely represents formation of a thin layer of inflammatory reaction with fibroblastic proliferation around the hematoma (Fig 63). Aneurysms due to injury may involve virtually any artery, including the aorta 6, 52, 108, 112, 113

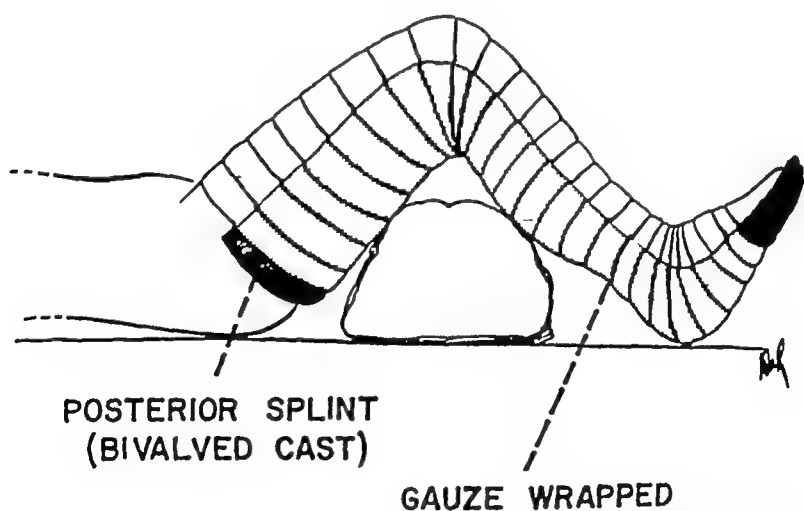


FIG 64 The extent of an arterial defect due to tissue loss can often be reduced by flexion of the extremity. This is especially true when the gaps involve the brachial and popliteal arteries. The flexion of the limb can be maintained postoperatively by means of a posterior splint

An arteriovenous (A V) fistula¹⁰⁷ is formed when the injury involves both the artery and the vein. As a rule simultaneous injury to both vessels has been sustained at the initial wounding but it would appear that occasionally a chronic aneurysm or pulsating hematoma of the artery may rupture into the adjoining vein and thus produce a true arteriovenous fistula. One mistake commonly made is to refer to an arteriovenous fistula as an arteriovenous aneurysm. Al

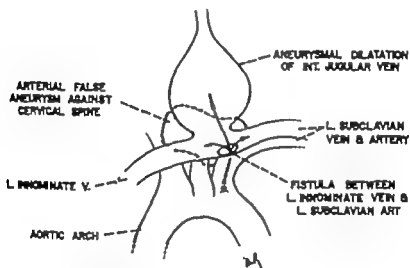


FIG 65 The anatomic changes indicated are often present in association with traumatic A V fistulae. There is venous dilatation at times arterial dilatation and not infrequently a false aneurysm. The illustration depicts the findings exhibited by a patient who was admitted with intractable heart failure and evidence of systemic infection possibly secondary to the fistula. The lesion was treated by rapid quadruple ligation and excision due to the very poor state of cardiac function the blood pressure being intermittently at shock levels. The marked cardiac enlargement present at the time of operation had subsided when she was re-examined six months later (see Fig 74)

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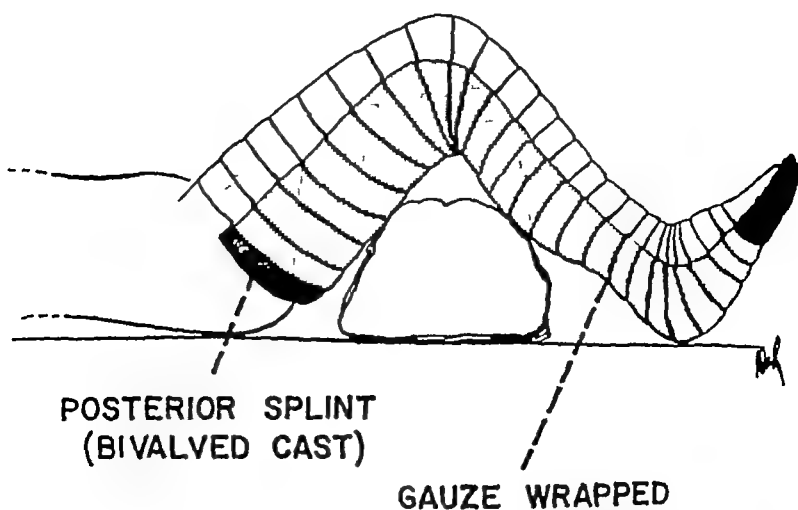


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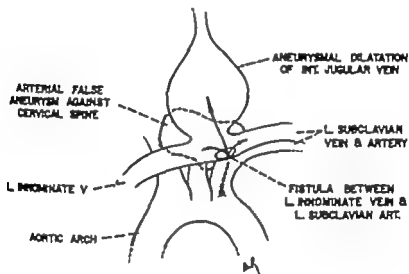


FIG. 63 The anatomic changes indicated are often present in association with traumatic A V fistulas. There is venous dilatation at times arterial dilatation and not infrequently a false aneurysm. The illustration depicts the findings exhibited by a patient who was admitted with intractable heart failure and evidence of systemic infection possibly secondary to the fistula. The lesion was treated by rapid quadruple ligation and excision due to the very poor state of cardiac function the blood pressure being intermittently at shock levels. The marked cardiac enlargement present at the time of operation had subsided when she was re-examined six months later (see Fig. 74).

though this designation of the lesion has the dubious merit of being time-honored, it is misleading to those not familiar with the pathology involved. It would be far better to refer to the lesion as an arteriovenous fistula, with or without associated aneurysm. For it is true that from time to time there is present not only a fistula between the artery and the vein but also a false aneurysm (Fig 65). Furthermore, marked dilatation of the artery proximal to an A-V fistula may in effect constitute a true aneurysm. When a knife or bullet passes through both artery and vein, the defect in the wall of the artery opposite the vein may bleed to form a false aneurysm or pulsating hematoma. The wall of the artery adjacent to the vein becomes fused to the vein and the communication between them is kept open by the force of the arterial blood rushing into the venous system.

The continuous bruit or hum caused by an arteriovenous fistula may develop within hours following the injury, or it may not develop for weeks, months or years. We treated one man whose local physician had detected a thrill over the site of a pistol wound within an hour following the infliction of the injury. In contrast, we treated one patient with a large arteriovenous fistula between the left subclavian artery and the left innominate vein which developed several years following the original stab wound. The patient first became aware of the presence of the lesion because of a sensation of "humming or buzzing" in the neck surrounding the fistula (Fig 65).

Causalgia, while representing a symptom rather than a structural defect, can be one of the major problems associated with traumatic injuries to nerves.

or arteries.¹⁰¹ This condition was first described by S. Weir Mitchell^{98, 99} and it has never been fully explained. Nevertheless the injury to a nerve or an artery, neither of which need be severed, results in a fairly characteristic type of reflex pain which appears to be mediated over the sympathetic nerve fibers to the extremity. Excessive sweating, erythema or pallor and trophic changes are usually observed in the part.

DIAGNOSIS OF ARTERIAL INJURY

The diagnosis of arterial injury is made by means of the history, the physical examination which may include blood pressure readings or oscillometry, arteriography where needed and surgical exploration in selected cases.

Any history of a knife or gunshot wound should alert one to the possibility of injury to major blood vessels as well as to other structures situated in the region concerned. Wounds of the chest or the abdomen which injure major arteries will usually be associated with sufficient hemorrhage to require prompt operation to prevent exsanguination if indeed the patient lives to reach the hospital. On the other hand should an artery in an extremity be injured any of a wide variety of clinical pictures may be exhibited by the patient at the time of examination. The wound in the artery may have clotted fairly promptly and aside from a hematoma of variable size surrounding the injured vessel little else may be noted at the time of admission. Assuming that swelling of the extremity is not marked and thus does not indicate massive blood loss within the wound any one of several possibilities may develop within hours or

days First, the artery may be divided, thrombosed or in severe spasm with no pulses present distally Second, injury to the wall of the artery may have been minor and it may heal entirely without sequelae Third, a pulsating hematoma may enlarge and become obvious due to secondary hemorrhage or to simple expansion of the false sac during the next few days A systolic bruit may be heard over false aneurysms from time to time Fourth, the patient may develop a thrill and continuous bruit over the site of injury within days or weeks, indicating development of an arteriovenous fistula Fifth, hemorrhage may not be apparent but severe causalgic pain may persist Thus it is apparent that for accurate diagnosis the examiner must have in mind a variety of possible variations in the clinical picture presented by a patient who has sustained an injury to a major artery

Significance of Pulses One common error is to interpret the presence of pulsations in the artery distal to the site of the possible injury as indicating that arterial injury has not been sustained This is of course erroneous for a number of reasons To begin with, if neither thrombosis, severe spasm, nor actual division of the artery with retraction of its ends exists, there is no reason why pulses would not be present distal to the level of injury Certainly occlusion of the profunda branch of the femoral or of the brachial artery is associated with a normal pulse at the foot or wrist (Fig 66) Furthermore, injury to the main arterial trunk with a false aneurysm or with an arteriovenous fistula is associated with pulses in the extremity distally Incidentally, use of oscillometer or even sphygmomanometer may assist in detection of differences in arterial pressure in paired members such

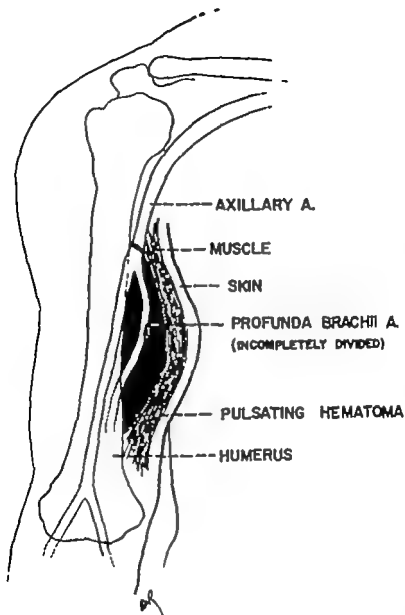


FIG 66 It may be seen that a false aneurysm (pulsating hematoma) arising from the deep brachial artery can be associated with normal pulses at the wrist. The same could be true of a false aneurysm affecting the main brachial artery so long as the artery was not thrombosed.

as arms or legs. Simple palpation is notoriously misleading in detecting slight to moderate differences in arterial pressure, and direct intra-arterial pressure measurements using a strain gauge will unquestionably be used more widely in the future.

The presence of a history of severe blood loss at the time of injury and while the patient was being transported to the hospital suggests major arterial injury. Rapid massive swelling of the injured part, before inflammatory edema of such magnitude would have accumulated, also suggests arterial injury. Most gunshot wounds of an extremity are eventually associated with considerable swelling, but this does not develop immediately. It is to be remembered also that severe blood loss is more likely to be due to arterial than to venous injury, since the venous pressure is not sufficient to overcome the resistance of surrounding tissues and therefore venous occlusion with clotting eventually occurs. Surprisingly little bleeding may occur when the artery is completely severed so that the ends can retract and provide mechanical assistance to hemostasis. We had one patient who had lost very little blood following a knife wound of the popliteal space, but the absence of pulses in the foot associated with a cold lower leg and foot prompted exploration of the injury. The ends of the completely severed popliteal artery were found several centimeters apart. Successful anastomosis was achieved with immediate restoration of foot pulses. Arteriography in such cases will often disclose extravasation of the radiopaque medium at the point of injury.

It has been emphasized that the presence of pulses distal to the site of entry does not exclude major ar-

terial defects. On the other hand neither does the absence of pulses distal to an injury establish the presence of thrombosis or loss of arterial continuity. Severe arteriospasm is a common finding in association with arterial trauma and many observers feel that it can be so severe as to produce gangrene. Such constriction can be verified by either direct inspection or arteriography and it can often be relieved by the local application of papaverine^{21 22} or by regional sympathetic nerve block. Thus when coldness pallor ischemic pain and sensory and motor changes in the extremity indicate marked ischemia paravertebral sympathetic nerve block may be indicated for a brief period of trial to demonstrate whether or not simple spasm exists or whether thrombosis or interruption of arterial continuity has occurred. If serious doubt still persists after a satisfactory block and arteriography is for one reason or another equivocal it will in many instances be desirable to explore the wound to exclude arterial injury. While an arteriogram may disclose extravasation of radiopaque medium into the tissues through a defect in the arterial wall at other times this study can be misleading regarding the level of arterial injury because of spasm or thrombosis²³. Nevertheless the use of the arteriogram in establishing arterial continuity and patency in the presence of arterial spasm has been helpful to us on a number of occasions. Should the pulse be absent on admission but return subsequently one still cannot exclude structural danger for the development of a pulsating hematoma (false aneurysm) days later may betray loss of arterial wall continuity. Again surgical exploration is highly desirable where

doubt exists regarding the integrity of an artery, particularly if ischemia in the extremity distally appears to be threatening its viability. Most gunshot injuries should be explored anyway to permit thorough debridement of devitalized tissue. Knife wounds need not be explored with such frequency unless there is evidence of nerve or blood vessel damage. When pulses were absent in the extremity distal to the site of a knife wound, we have usually found the artery to have been at least partially divided.

MANAGEMENT OF ARTERIAL INJURIES

General Considerations ^{19, 37, 70, 81, 90, 100}

The first requirement in the presence of a major arterial injury is to stop hemorrhage and to restore an adequate circulating blood volume. Unless massive bleeding can be controlled, it will be very difficult to stabilize the circulation. Point pressure firmly applied over the site of injury commonly suffices to stop the bleeding. This may be achieved by placing folded gauze dressings immediately over the wound and then encircling the leg with an elastic bandage. While less desirable and in certain respects actually hazardous, a tourniquet may be applied when point pressure does not suffice for the control of hemorrhage in an extremity. Of course, if the subclavian or the external iliac artery has been severed, it will not be possible to control bleeding with the use of a tourniquet. Under these circumstances one must infuse blood rapidly under pressure and operate immediately to gain control of the vessel. It should be emphasized that when much blood is being lost from a major artery it is absolutely essential to have available a means of pump

ing in blood under pressure. The methods of gravity flow, the use of syringes or the hopelessly inadequate squeezing of a small plastic cylinder interposed in the tubing can be pathetically lacking in such an emergency. An apparatus is now commercially available which permits one to exert air pressure in a bag surrounding the plastic bag containing the blood†. In this way the hazard of insufflation of air (with air embolism) is avoided while the advantage of pressure infusion is maintained.

If it has been possible to control bleeding it is preferable to delay operation until circulation has been reasonably well stabilized by adequate blood replacement. Only a short while is usually required if simple massive blood loss is rapidly replaced. However if a considerable period of time has elapsed since the massive blood loss began restoration of circulatory stability will not be as rapidly achieved even though normal blood volume is restored. Naturally the sooner operation is performed the less extensive will be the hematoma which dissects along various tissue planes to establish an excellent culture medium for bacterial multiplication or to interfere with wound healing.

As for the incision to be used much will depend upon the site of injury. In general it is preferable to make the incision in the long axis of the artery so that it can be extended as far proximally and distally as desired.²⁸ For instance it may be found that thrombosis has occurred for a considerable distance along the artery and removal of this thrombus is facilitated by an incision of adequate length. Local or systemic heparinization may or may not be employed.

† Manufactured by American Sterilizer Company

depending upon the circumstances. Postoperative regional sympathetic nerve block may be helpful.⁴² Further technical requirements will be noted in connection with the management of specific injuries now to be considered.

Specific Arterial Injuries

Contusion With or Without Spasm and/or Thrombosis Contusion of an artery may be produced in many ways. As mentioned above, the more common ones are blunt trauma due to compression, gunshot injury which may not actually strike the artery but which causes blast effect in the neighborhood of the vessel (Fig 63), the use of crutches which may damage the axillary artery if the pressure of the body weight is borne on the top of the crutch,^{67, 68} by fragments of a fractured bone,³² or by anterior dislocation of the shoulder.^{10, 81} In a given instance this contusion of the vessel may not produce serious alterations in blood flow, but in another it may result in severe ischemic disturbances. Intramural *thrombosis* with a bulging of the intima into the lumen may obstruct flow in this way, or there may be intimaluminal thrombosis which may propagate in either direction (Fig 67). Prolonged segmental arteriospasm⁸⁴ may precede thrombosis in many instances, the reduced volume of flow contributing to clot formation.⁶⁶ In addition to contusion and spasm, sustained *compression* of a vessel may also result in distal ischemia. We had one patient with a fracture of both the tibia and fibula who had had good foot pulses in the morning but who in the afternoon had a cold, ischemic foot with no pulses palpable and with anesthesia and loss of motor power in the toes. At first it was believed that she probably

had sustained thrombosis of the arteries to the foot perhaps due to trauma at the site of fracture. However someone suggested that additional weight be added to the traction which had been applied by means of a wire passed through the os calcis and when this was done an overriding of the fragments was corrected and pulses were immediately restored to the foot. Obviously the arteries had been either kinked or compressed and this is an important consideration in the management of fractures. It has been pointed out that arterial injuries associated with fractures result in a greater amputation rate than do arterial injuries not associated with fractures.²⁰

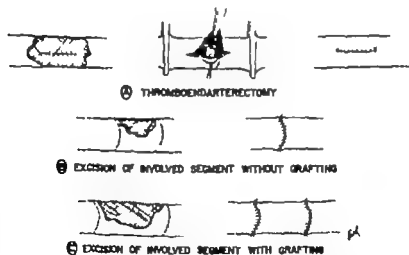


FIG. 67. A thrombosis can often be removed by simple arteriotomy but if the adjacent wall of the vessel is damaged the involved segment may have to be excised. The arteriotomy incision may be either longitudinally or transversely. Following excision of an arterial segment a graft may be required to permit restoration of arterial continuity without undue tension.

Injury to the axillary artery following anterior dislocation of the shoulder has been emphasized^{10, 84} In one case reported by McKenzie and Sinclair⁸⁴ a tear in the wall of the subscapular artery at its point of origin from the axillary artery was apparent. Thrombosis at this site had extended into the axillary artery, resulting in an organized thrombus in this vessel. A one centimeter segment of contused axillary artery and additional clot were removed. The artery was mobilized and a primary end-to-end anastomosis performed with restoration of normal pulses at the wrist.

Arterial Spasm³ The importance of a tear in the wall of an artery, due to the laceration of a small collateral branch, was emphasized by Edwards and Lyons²⁶ They stressed the importance of hemorrhage into the perivascular sheath that may result in compression of the arterial lumen. In their series of cases it appeared that severe localized arteriospasm had eventually resulted in thrombosis. In one patient with serious ischemia only severe spasm was found, no thrombosis being demonstrated even by opening the artery at operation. These workers found that procaine produced an inadequate relief of arterial spasm when injected into the area, whereas a solution of papaverine when applied to the circumference of the artery by means of a moistened sponge gradually resulted in relaxation of the spasm over a period of several minutes.⁷² Care was taken to cover the most proximal extent of the spasm. The sponge was left in place for from 15 to 20 minutes and then removed, the spasm was relieved and was not again precipitated by manipulation of the artery. Similar success was obtained by Jahnke and Howard,⁶⁰ who used a sponge

soaked in 2.5 per cent papaverine hydrochloride solution. Sympathectomy may be helpful.¹¹

In discussing the problem of vascular injury without laceration Edwards and Lyons⁸ emphasized the frequency with which blunt trauma especially that associated with fractures, results in arterial spasm or thrombosis. Their experience gained through routine exploration of all injuries above the knee or elbow in which the peripheral pulse was markedly diminished or absent led them to discover various types of unanticipated lesions. In one case there was a tense hematoma beneath the perivascular fascia associated with marked segmental arteriospasm. An incision through the artery disclosed no thrombosis. The spasm was not relieved by procaine but was relieved by the application of papaverine solution. In another case severe arterial spasm was found to be associated with arterial thrombosis. The thrombus was removed but, because of contusion of the arterial wall a segment of the femoral artery six centimeters in length was resected and the defect bridged by a similar length of saphenous vein taken from the same leg and reversed before anastomosis. In a third case exploration of a femoral artery in the presence of absent distal pulses revealed an area of spasm two centimeters in length with ecchymosis beneath the adventitia. A segment of the contused artery was resected as recommended by Leriche¹² and was replaced with a reversed saphenous vein graft taken from the opposite leg. Pulses were restored in the foot. These and other similar cases in the literature attest the severe spasm which may accompany arterial injury and which may or may not be associated with thrombosis.

That traumatic vasospasm with its complications following trauma is not always an innocuous and fleeting problem has also been stressed by Gaidner⁴³ He reported that depending upon its severity, with or without thrombosis, it might produce Volkmann's ischemic contracture, necrosis of muscle bundles or even gangrene of the entire limb In one case he described there was Volkmann's ischemic necrosis of the muscles of the calf of the leg associated with severe arteriospasm, and in another there was a similar necrosis of muscles of the arm associated with arteriospasm that developed even in the absence of injury to the arm Even spinal anesthesia had failed to relieve arteriospasm under some circumstances It was emphasized that Volkmann's ischemic contracture⁴⁹ represents not a separate entity but merely a stage between the complete recovery of the limb and total gangrene, the muscles perishing because of their great need for oxygen The suggestion was made that in at least some cases vascular spasm appears to arise as a result of the reaction of the entire organism to emotional and metabolic disturbances, possibly mediated through the endocrine system His results in the management of Volkmann's ischemic contracture had been disappointing in a number of instances

Elliott³² reported a case of axillary-brachial arterial occlusion which had resulted from disruption and dislodgement of a portion of the intima of the brachial artery by fragments of a fracture of the humerus The associated thrombus and dislocated intimal fragment were removed, and arterial continuity and blood flow were successfully restored

Management of Arterial Thrombosis. Many con

considerations that are important in the management of arterial contusion with spasm and thrombosis have been mentioned already. In Figure 67 are shown the procedures that are frequently used. Simple contusion of the artery without thrombosis or spasm requires no surgical intervention if the pulse is restored either spontaneously or by regional sympathetic nerve block. If distal pulses cannot be restored the wound should be explored. Sub-adventitial hematomas should be evacuated and spasm relieved by the application of papaverine solution. If thrombosis exists the thrombus should be removed. The presence of retrograde flow following removal of clot, perhaps achieved by the retrograde flush technique,¹⁷ is a favorable prognostic sign. Successful restoration of blood flow has been established many hours following arterial occlusion. If necessary the involved segment of the artery should be resected. A defect or gap of from one to two centimeters in length can frequently be overcome by freeing up the artery proximally and distally and at times by suitable positioning of the extremity to provide the maximum in relaxation of tension on the vessel. If primary anastomosis cannot be performed without the use of a graft it may be inserted (Fig. 67). Saphenous vein grafts may be suitable where muscle support exists but otherwise some other prosthetic material should be employed. Heparin may or may not be used depending upon the circumstances; the wound is carefully debrided thoroughly irrigated and frequently drained particularly if the patient is to be heparinized. Postoperatively continuous spinal or caudal anesthesia or other regional sympathetic block may be employed if desired.

been injured the wound should be explored. It may be seen in Figure 66 that a large collateral branch can be either partially or completely severed with the continuation of pulsations distally and in such an instance the presence of such pulses would be of no assistance in excluding the possibility of arterial injury. Failure to explore such a wound at the time of admission may result in repeated episodes of secondary hemorrhage and inordinately prolonged disability when simple suturing or ligation could have been carried out immediately when the patient was first injured with prompt recovery. We know of one patient whose thigh was drained three separate times to evacuate hematoma before formal and definitive exploration of the depths of the wound by an experienced surgeon using adequate anesthesia and exposure disclosed a partial interruption of the deep branch of the femoral artery. All that was required was to ligate and divide the vessel and to evacuate the infected hematoma. Thereafter the patient was discharged in less than a week though it had been almost six weeks since she was originally injured. Arteriograms were not so commonly used then but such a study would probably have disclosed the pulsating hematoma (false aneurysm) in her case.

Treatment. When incomplete or complete division of an artery appears likely suitable anesthesia is employed to permit adequate exploration of the wound. The first step as with all types of arterial surgery is to gain control of the vessel above and below the point of probable injury. Following this one explores the tract to identify not only whether or not arterial injury has been sustained but also whether or not venous



EXCISION OF DAMAGED
PORTION AND LATERAL
SUTURE OF DEFECT

(A)

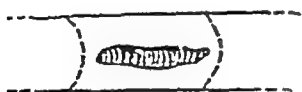


EXCISION OF DEFECT
WITH PRIMARY ANASTOMOSIS

(B)

(C)

EXCISION OF DEFECT
WITH GRAFTING



(D)

DEBRIDEMENT, CLOSURE
OF LONGITUDINAL TEAR

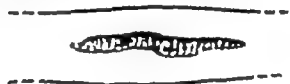


FIG. 68 Although some authors recommend routine excision of the lacerated segment of an artery, the writer has been satisfied with a V type excision of a small defect with transverse closure (A) in selected cases. Other lacerations will require the excision of a segment of the vessel with or without grafting. Longitudinal closure of a laceration

and nerve lesions are present. Necrotic muscle will commonly be found following gunshot injuries but knife wounds require relatively little debridement except for the removal of hematoma. The site of injury of the vessel is inspected. Optimally there should be brisk bleeding from the proximal end and at least some back bleeding from the distal end. Any thrombus should be removed from each end of the vessel by appropriate maneuvers; these may include stripping of the vessel between two fingers or suction wrapping the leg with an elastic bandage from below upward or the retrograde flush technic by introduction of a cannula into the posterior tibial artery through which saline is flushed in a retrograde direction in the instance of a leg wound. The thrombus in the proximal portion of the artery will usually be expelled promptly by the force of the arterial stream particularly when the clot just inside this vessel has been removed. As a matter of fact one will thereafter be forced to apply clamps or tapes to prevent excessive blood loss from the spurting proximal end of the vessel. The distal end of the vessel often entails a more difficult problem since the pressure in this portion of the artery is low and clotting not only of the main trunk but also of collateral vessels may have occurred; this thrombus may be hard to extricate. Once the artery has been cleansed of intraluminal clot and the divided ends or the defect producing incomplete division has been freshened (or more extensively debrided in the presence of gunshot wounds) a simple transverse closure or an anastomosis is performed with fine arterial silk (Fig. 68). In the adult patient a con-

whose margins must be debrided (D) is not recommended since narrowing of the lumen will usually result.

tinuous over and over on baseball type of stitch is satisfactory, but in younger individuals the suture line should be interrupted on at least one side to permit growth of the arterial lumen with increasing age. It has been shown that a continuous suture line allows less enlargement of the lumen with growth than does an interrupted suture line.⁶⁰ It is most important to remove all intraluminal clot just prior to completion of the anastomosis. The wound is then thoroughly irrigated and a drain inserted if indicated.

While arterial spasm may reduce the volume of arterial pulsations in the foot immediately following surgery, it has been our experience that a technically satisfactory anastomosis is usually rewarded with distal pulsations immediately. If these are absent, the possibility of thrombosis at the site of the anastomosis is a real one. Any arteriospasm can often be relieved with papaverine solution (2.5 per cent) applied locally, and this should be done before the wound is closed—for a good volume of flow constitutes the best possible protection against postoperative thrombosis at the site of anastomosis. We have not commonly performed sympathectomy or employed regional sympathetic nerve block postoperatively.

True and False Aneurysm

The true and false aneurysms which follow arterial trauma will be considered together since they are managed in more or less the same manner (Fig. 72). These lesions too result from injury to the arterial wall,⁶¹ but they may not develop or become prominent for days or weeks. A true aneurysm results from damage to the arterial wall which permits a bulging or dilata-

tion at that point but prior to late rupture some portion of the wall remains intact at all points (Fig 68). A false aneurysm or pulsating hematoma in contrast forms at a point where arterial continuity has been lost such as is shown in Figure 69. True aneurysms were dealt with in Chapter 5 and will not be considered in detail here.

False Aneurysms. The anatomic defect which permits development of a false aneurysm is illustrated in Figure 68. A photograph of such a lesion which resulted from a laceration by a broken bottle is shown in Figure 69. Initially the bleeding had presently ceased following application of a pressure dressing, but in the ensuing weeks hemorrhage from the depths of the wound had occurred repeatedly. Thus chronically recurring hematoma plus granulation tissue had



FIG. 69 *False Aneurysm* Patient had cut her wrist on a broken bottle several weeks previously this pulsating hematoma developed due to a defect in the ulnar artery (see text)

tinuous over and over on but satisfactory, but in younger individuals should be interrupted on at least the growth of the arterial lumen. It has been shown that a continuous enlargement of the lumen with an interrupted suture line ⁶⁰ does not remove all intraluminal clot just proximal of the anastomosis. The wound is irrigated and a drain inserted in the

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True and False Aneurysms

The true and false aneurysms which develop after trauma will be considered together. In most cases the lesions are more or less the same. In most cases the lesions too result from injury to the arterial wall but they may not develop or become apparent for days or weeks. A true aneurysm is a localized dilatation of the arterial wall which permits a

the repair of such small vessels is readily feasible but we would not hesitate to do so. It is to be remembered that either the radial or the ulnar arch is absent in perhaps 5 per cent of people and thus one should not unnecessarily sacrifice either artery without being reasonably certain that the other is present. Even within the past year we have treated an injury in which bromsulphalein was inadvertently injected into the radial artery instead of into a vein followed by severe burning pain in the hand and eventual gangrene of the thumb and a portion of the adjacent two fingers. It is possible that this patient does not have an ulnar arch.

It may be seen that there is frequently an inordinate delay in the accurate diagnosis of false aneurysms especially since pulses are often present in the more distal portion of the extremity. Attention is again called to Figure 66. It may be seen that when the hematoma lies deep within the tissues of an extremity it may be difficult indeed to determine precisely what the arterial lesion is and even that one exists especially when the thigh is involved. Arteriograms should be used far more frequently than they have been used in the past for the precise identification of anatomic defects secondary to traumatic injuries.

The excision of a chronic false aneurysm is shown in Figure 70. The patient had been stabbed in the thigh several weeks previously. Following correction of the arterial defect the hematoma should be removed as completely as possible since infection in such wounds is not uncommon. When an anastomosis must be performed in the presence of contamination there is always a serious hazard that the suture



FIG. 70 *False Aneurysm* Top This pulsating hematoma involving the left femoral artery followed a stab wound. However, since all pulses had been present immediately following injury, the arterial damage was not suspected until a pulsating mass developed later. Bottom

line may break down with secondary hemorrhage. Furthermore there will be slight probability of a successful secondary anastomosis in an infected field. We know of a recent case in which a false aneurysm was resected with the interposition of a graft in the femoral artery. Thereafter staphylococcal infection developed in the wound and the graft broke down with severe secondary hemorrhage that almost cost the patient his life. Pressure was applied to the extremity until the patient could be taken to the operating room following which the artery was ligated both above and below the site of previous interposition of the prosthesis. Unfortunately distal ischemia of the limb was so great that after many weeks the limb was amputated because of ischemic muscle necrosis and contracture and failure of skin healing.

Traumatic Aortic Aneurysms. Although discussion thus far has centered around false aneurysms involving the extremities, traumatic rupture of the aorta^{89 78 101} with pulsating hematomas¹⁰⁴ may occur within the abdomen or thorax as well^{11 46 47 89}. In discussing surgical management of rapidly expanding intrathoracic pulsating hematomas involving the aorta Shumacker and King¹⁰⁶ urged that in all thoracic injuries every effort be made to determine as early as possible any evidence of aortic rupture. They stressed the hazard of sudden massive hemorrhage when a direct thoracic approach is used and they proposed initial interpolation of a graft between the ascending aorta and the descending thoracic aorta to provide additional safety a procedure which they had used. This permits oc-

A 1 cm. segment of the artery was excised and arterial continuity restored using interrupted sutures.

clusion of the aorta at some distance proximal and distal to the false aneurysm prior to dissection in the vicinity of the aneurysm itself

The *roentgen diagnosis* of acute and chronic traumatic aneurysms of the thoracic aorta ¹²¹ was reviewed by Steinberg and Evans ¹¹³ They emphasized that acute traumatic aortic rupture associated with fractures, internal injuries and shock is often overlooked, that an awareness of the role of stresses incurred in deceleration following blunt trauma, as is common in automobile or airplane accidents,⁹⁸ should direct attention to the arch of the aorta just beyond the origin of the left subclavian artery, since this is the most common site of rupture of the aorta in such accidents (Fig 71) The aorta is relatively fixed at this point

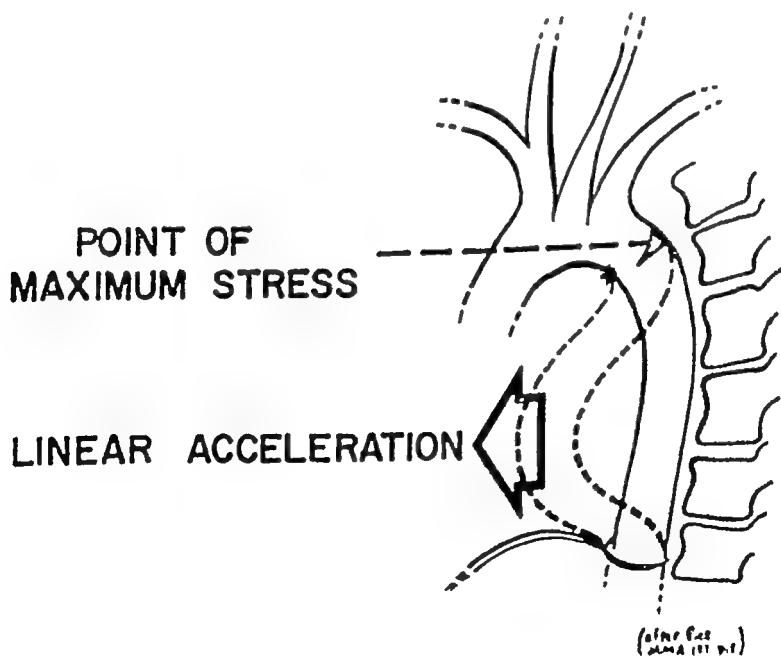


FIGURE 71

but may be arched anteriorly below avulsing intercostal arteries. These authors recommended that serial roentgenograms of the chest be made for many weeks following injury to permit recognition of mediastinal hematomas hemothorax or the development of a localized bulge in the region of the aortic knob representing the aneurysm. It was pointed out that unless chest roentgenograms are made routinely after thoracic injury the development of the traumatic aneurysm may not be appreciated for a number of years. Angiocardiography is valuable.

It has been found at autopsy that patients may sustain completely asymptomatic traumatic aneurysms of the aorta.⁸⁸⁻¹⁰¹ Chronic lesions are diagnosed on the basis of the above findings plus fluoroscopy esophagrams the characteristic location of traumatic aneurysms just distal to the origin of the left subclavian artery the absence of positive serologic tests for syphilis and the history of trauma. A review of 72 cases of traumatic rupture of the aorta and 24 cases of spontaneous rupture was published by Strissman.¹¹⁴ The chief differences regarding the occurrence origin and pathologic picture of both lesions were examined.

Management.²⁷⁻³¹ Few repairs of true traumatic aneurysms of the thoracic aorta carried out shortly following the accident have been reported. The patients are usually in very serious general condition and frequently the aneurysm may not develop or be diagnosed for some time. In contrast numerous subacute or chronic traumatic aneurysms of the aorta have been resected and grafted some with the aid of extracorporeal bypass. Gerbode and associates⁴¹ re

both cases that he reported. In 1861 Broca¹² recorded a remarkable instance of proximal dilatation of the artery. Despite a marked distention and thinning of the wall of the axillary and brachial components proximal to the fistula, the artery abruptly returned to normal size immediately below the level of the arteriovenous fistula. Although Halsted had in 1918 called attention to the frequent association of cardiac enlargement with arteriovenous fistulas and had surveyed world literature on the subject,¹⁷ which was later

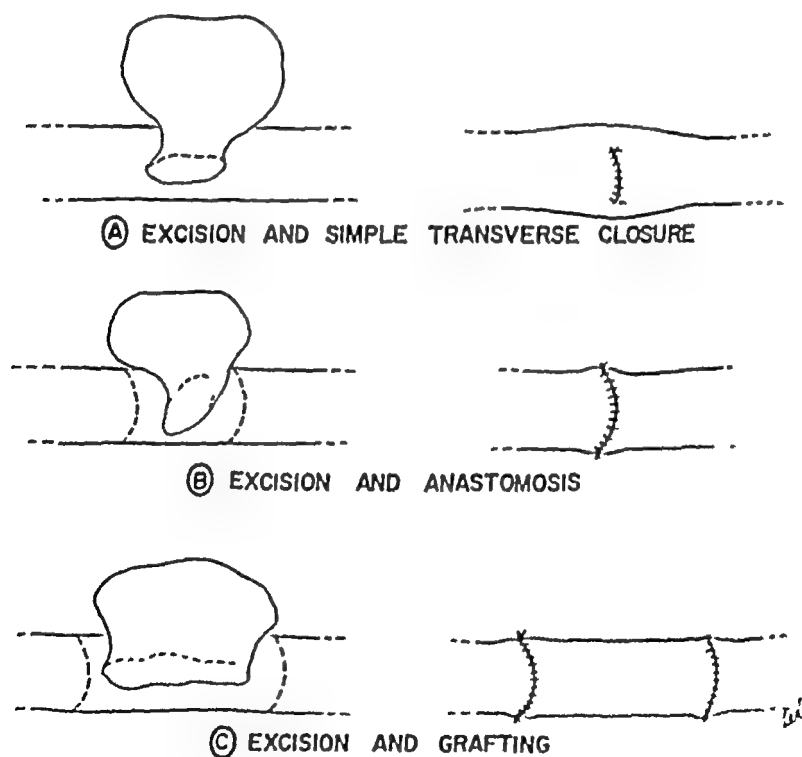


FIG. 72. False aneurysms which arise from a small arterial defect can often be managed by simple excision with transverse closure following freshening of margins of defect (A). Larger segments of defective vessel are best managed by segmental excision, with or without grafting (B, C).

published by Callander¹² to Emile Holman¹⁷ must be accorded special credit for his studies in 1923 and subsequently which established that arteriovenous fistulas may produce dilatation of the artery and cardiac enlargement. He constructed femoral arteriovenous fistulas in dogs and demonstrated classic alterations in blood pressure and pulse rate which occur following construction of such fistulas. These alterations could be immediately abolished by firm digital pressure over the site of the fistula. He attributed slowing of pulse rate upon occlusion of the fistula to distention of the aorta by blood with resultant slowing of the heart by vagal depressor fibers in the wall of the aorta (and possibly by reflex changes arising in the carotid sinuses).

Types and Sites of Arteriovenous Fistulas^{18, 17, 117}

Although emphasis here is placed upon traumatic arteriovenous fistulas, it should be borne in mind that various other etiologic circumstances may give rise to shunting of blood from arteries immediately into veins without passage through intervening capillaries. Rupture of an atherosclerotic aneurysm or plaque into an adjacent vein may cause such a fistula¹¹. Congenital arteriovenous fistulas are represented by patent ductus arteriosus, cavernous hemangiomas, pulmonary arteriovenous fistulas and normal fistulas that open up under physiologic circumstances. Infection may also produce arteriovenous fistulas.

Traumatic arteriovenous fistulas may occur anywhere that an artery and a vein lie in close approximation to each other including the aorta and vena cava^{11, 4, 11}. In Figure 73 are shown the locations of 12 arteriovenous fistulas treated in the Hospital of the University of Mississippi over the past four years.

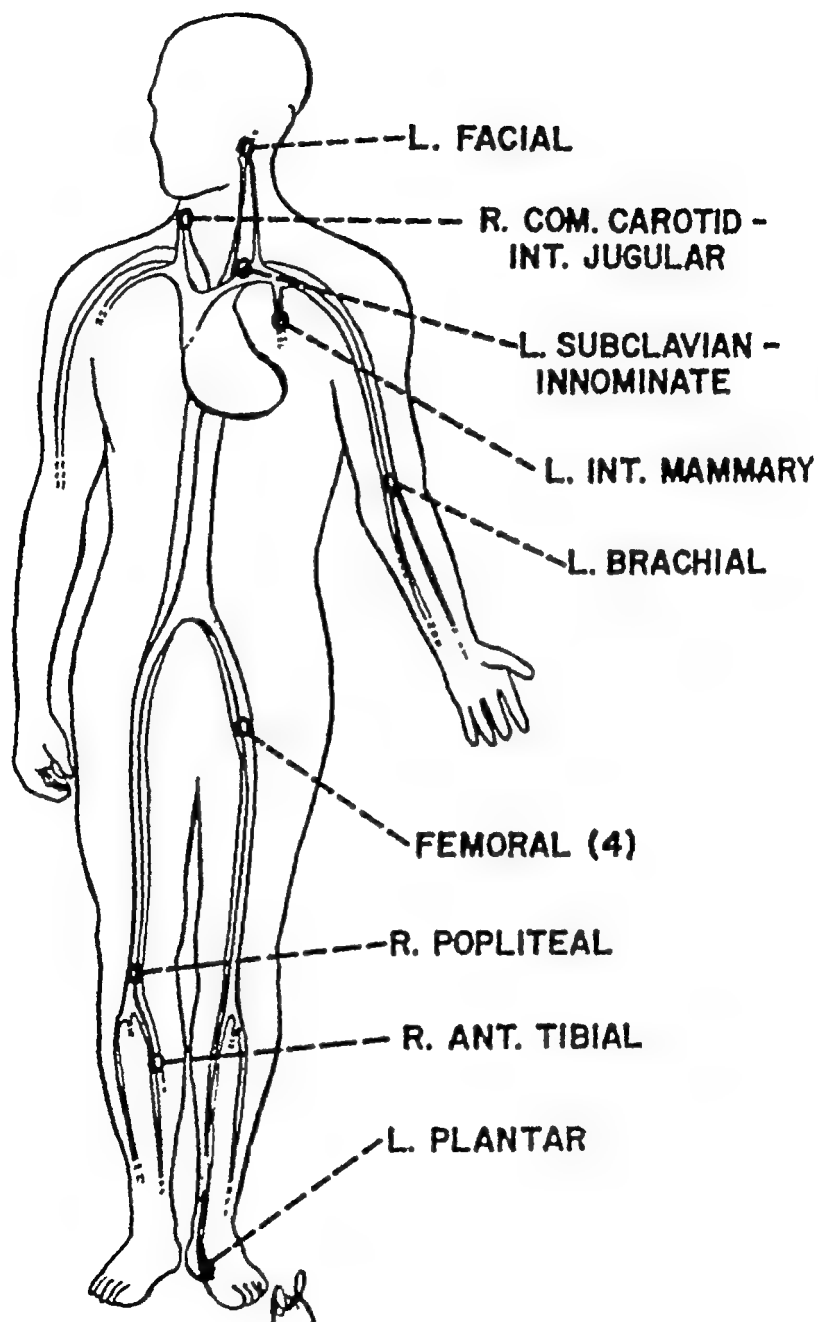


FIG 73 The writer participated in the management of the 12 fistulas shown above over a four year period

Most of these fistulas were the result of either knife or gunshot wounds and the large majority were treated by direct surgical repair shortly following their occurrence or discovery. Serious cardiac enlargement (Fig 74) and evidence of heart failure were present in several of these patients who had large fistulas that had been present for a number of months or years.



FIG. 74 Left. Cardiac enlargement due to chronic A V fistula (see Fig 65) Right. Normal cardiac silhouette six months following excision of the fistula

Pathophysiology of Large Traumatic Arteriovenous Fistulas.^{21 22 23} *Early Changes* Immediately following injury by knife or missile there may be only the local evidence of trauma without evidence of an arteriovenous fistula. Yet in one patient we treated, the fistula was obvious to his local physician almost immediately following an accidental bullet wound of the thigh. In most instances however the presence of the characteristic thrill and bruit, present throughout the cardiac cycle usually is not detected for days

weeks, or months. It is probable that many fistulas enlarge and thus become more prominent, whereas some small fistulas may close spontaneously.⁶³

Once the arteriovenous fistula has been well established, as between a femoral artery and a femoral vein, the increase in venous blood flow surrounding the lesion may result in venous distention and an increased warmth of the overlying skin. Often there is swelling due to distention and edema surrounding the fistula, and not uncommonly the patient himself will have noted a "buzzing" sensation. This is especially noticeable to the subject when the arteriovenous fistula is situated in the neck, as between a carotid artery and an internal jugular vein. The short-circuiting of the usual volume of flow to an extremity distal to a fistula may result in relative ischemia of the part. This may even cause rest pain, but it is usually reflected more prominently in intermittent claudication on exercise.

When the arteriovenous fistula is discovered, the thrill and bruit can be obliterated by pressure firmly applied over the site of the fistula if it is accessible. Moreover, as noted above, if the pulse rate and blood pressure be taken immediately prior to the obliteration of the bruit by pressure over a large fistula, there will result a slowing of the heart rate and a fall in the systolic blood pressure associated with a rise in the diastolic pressure—that is, a narrowing of the pulse pressure. Further confirmation of the presence of an arteriovenous fistula may be achieved by demonstrating an increased venous pressure and an increased oxygen saturation of blood in the distended veins sur-

rounding the fistula. With arteriography the defect itself may be outlined.

Late Changes Eventually a large arteriovenous fistula may result in a number of well-documented changes. First total blood volume will increase. Epstein and Ferguson²³ found that this increase in total blood volume was due almost entirely to an expansion of the plasma volume with no change in total red cell mass. It has been mentioned previously that cardiac enlargement frequently occurs and less commonly in our experience, there may be dilatation of the artery proximal to the site of the fistula. The wall of the veins surrounding the fistula may become thickened at times referred to as arterIALIZATION of these veins. At operation for an arteriovenous fistula it may at times be difficult to determine what is vein and what is artery. In fact when quadruple ligation with excision was commonly practiced the pathologist could not always be certain of identity of vein *versus* artery.

Cardiac failure will often ensue if a large fistula is allowed to remain. Nevertheless the lapse of time between the creation of the arteriovenous fistula and the development of cardiac enlargement and heart failure varies considerably from patient to patient, even when the fistulas are of approximately the same size. Fistulas are compatible with a normal life of a great many years if they are small and Dorney²⁴ has reported the existence of such a fistula for 57 years. Cardiac enlargement usually recedes following closure of a fistula (Fig. 74).

Subacute Bacterial Endocarditis and Endarteritis. Subacute bacterial endocarditis and endarteritis occasionally develop in association with large arterio-

venous fistulas⁶¹ In 1935 Hamman and Rienhoff⁶² reported a case of *Streptococcus viridans* septicemia that was cured by the excision of a fistula between the iliac artery and vein In 1916 Cutler and Wolff⁶³ reported an acquired arteriovenous fistula with coexistent subacute bacterial endocarditis and endarteritis And later Lillehei and his associates⁷⁸ demonstrated that subacute endocarditis could be produced experimentally in dogs by the construction of large bilateral arteriovenous fistulas

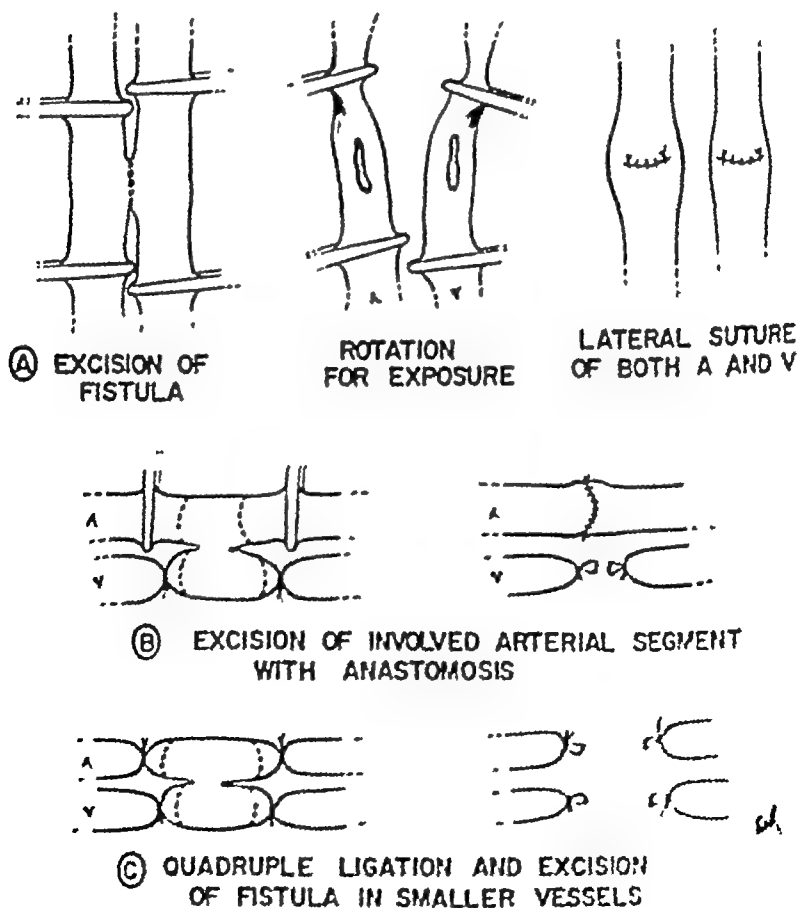


FIGURE 75

Management of Traumatic Arteriovenous Fistulas.²⁴

^{24, 24 192} Management of traumatic arteriovenous fistulas (Fig 75) has been revised since the close of World War II. Prior to that time most arteriovenous fistulas were allowed to become chronic, where possible and then were excised by quadruple ligation and excision.²⁵ In a large percentage of cases this resulted in subsequent relative ischemia of the extremity distal to the site of excision of the fistula,² regardless of the length of time which had been permitted for collateral circulation to develop to diminish the risk of gangrene following excision of the fistula. In some cases it was of course not possible to allow several weeks or months to go by with the objective of permitting the establishment of increased amounts of collateral circulation since either heart failure from a large fistula or more frequently risk of hemorrhage from an associated enlarging false aneurysm had to be considered and operation performed promptly.

In marked contrast to the practice of quadruple ligation and excision the previously reviewed experience in the Korean War with arterial repair has revolutionized management of not only simple arterial injury but also of arteriovenous fistulas. It has been found that in management of almost all arteriovenous fistulas it is possible to re-establish arterial continuity and in perhaps a third of the cases to re-establish venous continuity as well following taking down of the fistula. Our personal experience has been quite similar. During the past few years we have operated upon arteriovenous fistulas as soon as they were discovered. By identification of the vein and the artery both above and below the site of the fistula it is possible to approach the fistula by sharp dissection and to take it

down We have not invariably completely transected the artery Rather, when the defect in the arterial wall has been small we have simply excised this portion using a V-shape type of excision (Fig 68) At other times, where a considerable portion of the circumference of the wall of the vessel was involved, this segment of the artery has been sharply excised and a primary anastomosis of the artery performed To do this it has occasionally been necessary to free up the artery both above and below for a considerable distance, perhaps even dividing one or two collateral vessels and positioning the extremity in such a manner as to achieve maximum relaxation of the artery Such a flexed position of the extremity should be maintained for from seven to ten days postoperatively using a plaster splint It is almost invariably possible to restore arterial continuity and, frequently, venous continuity as well by a similar type of operation upon the vein A graft may be used if required ⁴⁵

Complications of Repair of Arteriovenous Fistulas
Although complications of the repair of arteriovenous fistulas are relatively few, several deserve mention First, it is of course obvious that one must avoid damage to nerves which may be incased in the hard fibrous reaction which surrounds a chronic arteriovenous fistula This protection of the nerve from injury is best achieved by freeing up the nerve both above and below the level of the arteriovenous fistula and then tracing it very carefully into the fibrous or scar tissue, maintaining identification of the structure until it can be fully separated and retracted A second complication of arteriovenous fistula is to have the fistula recur However, one suspects that frequently it is not

recurrence but the presence of a second fistula whose bruit could not be detected until the first fistula had been closed that accounts for the so-called recurrence. We had such an experience in operating for an arteriovenous fistula in the thigh of a medical student injured in a duck hunting accident. To our intense dismay several days following operation the resident detected a bruit at a distance from the site at which an arteriovenous fistula between a large muscle branch of the femoral artery and an adjacent vein had been excised. Prompt reoperation disclosed a second smaller arteriovenous fistula between another large muscle branch of the femoral artery and an adjacent vein. In another patient injured by a shotgun accident there was one arteriovenous fistula in the right antecubital fossa and a second one in the left popliteal fossa. Both of these were managed at the same operation. Since the experience with the medical student, we have employed a *sterile stethoscope* at operation both to identify rapidly and precisely the exact site of the fistula when palpation is less strong than usual and to avoid missing a second lesion.

Third thrombosis may occur at the site of arterial anastomosis or repair but this is not common. We have not frequently employed heparinization since we have found from experience that careful and meticulous arterial anastomosis with minimal trauma and fine suture material will be followed by a satisfactory distal pulse in a high percentage of cases. In fact, instances of hemorrhage following the use of heparin have somewhat reduced our enthusiasm for its use systemically in patients who have sustained trauma. Our usual procedure is to explore and re

was restored but the vein ligated. This prompted a policy of venous repair wherever possible, and it was found that this could be accomplished in approximately 30 per cent of the major veins involved in arteriovenous fistulas. Incidentally, eight patients required no operative intervention, since spontaneous closure of their fistulas and aneurysms occurred during hospitalization. Six of these involved major vessels such as the common carotid, axillary and popliteal arteries. Two of the patients with arteriovenous fistulas of the iliac vessels had developed these complications following lumbar intervertebral disc surgery, a complication now generally appreciated ^{46, 51, 58, 79}

Hughes and Jahnke ⁶³ found that obliterative techniques were acceptable for most minor vessel lesions, but that 50 per cent of patients who underwent obliterative procedures for lesions of major vessels had poor results. Sympathectomy was found to be a poor substitute for reparative vascular surgery, with which everyone would agree. The presence of late varicosities and swelling of the extremities appeared to be related to venous ligation. It was concluded that the results of reparative operation for arteriovenous fistulas were excellent, but that every effort should be made to repair both the artery and the vein to prevent late swelling of the leg.

Causalgia

This discussion of arterial trauma would be incomplete without further mention of causalgia ^{8, 22, 23, 25, 83, 116}. This is a post-traumatic pain syndrome which may follow various types of injuries, particularly those involving arteries or nerves. The precise anatomic

pathways over which this pain is experienced are debated and in fact may be variable but the sympathetic nervous system has an intimate relationship to the pain in many cases.²¹⁻²³ In fact most observers feel that the majority of instances of causalgic pain should be relieved by blockade or section of the sympathetic nerve supply to the part involved.²⁴⁻²⁵ Typically the patient has a burning pain atrophy of the skin excessive sweating and hyperesthesia. Touching of the involved extremity or even jarring the bed may at times suffice to set off severe pain when the patient was previously comfortable. The condition can be most debilitating and it may be extremely chronic. On the other hand procaine block of the sympathetic nerves supplying the part even once may suffice to break the reflex pathways and the causalgia may not recur. In other circumstances the procaine block establishes the fact that the pain is affected by blockade of the sympathetic nerve fibers to the part,^{21,26} and sympathectomy may then relieve the condition permanently. Nevertheless, not always is the pain relieved by division of the sympathetic nerve fibers though frequently there remains some doubt as to whether the sympathectomy was complete.

Closely related to the serious causalgias are traumatic edema and osteoporosis. Sudeck's atrophy of bone reflex dystrophy of the extremities²⁷ segmental arterial spasm and other conditions. Over the years there has been a tendency to divide the various instances of causalgia into so-called minor and major causalgias.²⁴⁻²⁶ Larsen²⁴ has pointed out that true major causalgias are rare in civilian practice, though minor causalgias are relatively common. The causal

gia which follows nerve injury seems to be more often associated with torn, crushed or partially lacerated nerves than with cleanly incised nerves. There has been some evidence that this is also true of arterial injuries, since frequently the artery need not have been severed or even partially divided for causalgia to occur. However, in most instances it is difficult to be certain that nerve injury is not also involved, since in the extremities the artery and the nerve frequently lie in close proximity to each other.

Treatment It is sufficient in many cases simply to place the part at rest, to avoid trauma to the involved extremity, to give appropriate sedation, and to allow time for the causalgic state to subside spontaneously. This process may be hastened by a blockade of the regional sympathetic nerves with procaine or xylocaine. In fact, as pointed out above, a single infiltration around the sympathetic nerves with a local anesthetic agent may suffice to break the reflex arc temporarily and this may prove to be permanent. Use of tranquilizing agents has been helpful in some instances.

Nevertheless, there are many cases of severe causalgia with the typical burning pain, skin atrophy, excessive sweating, and erythema of the part which do not respond to conservative measures. These patients are miserable and are virtually incapacitated by their condition until permanent relief is obtained by sympathectomy.⁷⁰ Although numerous cases are reported in which the causalgic type pain is not so relieved, in our experience it usually has been relieved by sympathetic nerve section. Even so, in many cases in which sufficient time has been allowed there has been a spontaneous regression of the symptoms. Again,

true causalgic type pain should be differentiated from other types of pain such as the shoulder arm hand syndrome which follows myocardial infarction and which may be associated with or a prelude to reflex muscular dystrophy of the involved arm usually the left. Whereas rest and passive physical therapy frequently will relieve the symptoms and physical findings of reflex dystrophy such measures have not proved to be as effective in the relief of causalgia.^{18, 40}

Summary and Conclusions

1 Arterial injuries often assume primary importance in the general management of trauma.

2 Historical background types diagnosis and treatment of traumatic arterial lesions are considered.

3 The problems resulting from arterial injury include contusion with or without spasm and/or thrombosis laceration which may represent incomplete or complete division loss of arterial substance aneurysms (true and false) arteriovenous fistula and causalgia.

4 The diagnosis of arterial damage is achieved by means of the history and physical examination and arteriography plus surgical exploration when indicated where doubt exists. The presence or absence of pulses distal to the site of injury neither establishes nor excludes the presence or the absence of organic arterial damage. Arteriography should be employed more frequently than it has been in the past.

5 Amputation rates following arterial injury have been greatly reduced by prompt arterial repair. However success achieved by direct arterial surgery is influenced by the presence and duration of shock, extent of

other soft tissue injuries and fractures, length of time elapsing prior to operation and infection

6 Most traumatic arteriovenous fistulas can be repaired by direct arterial reconstruction when discovered

7 Recent advances in the management of acute and chronic traumatic arterial lesions now permit restoration of arterial continuity and pulsatile flow in many military and perhaps most civilian injuries

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